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Illinois Medical Journal

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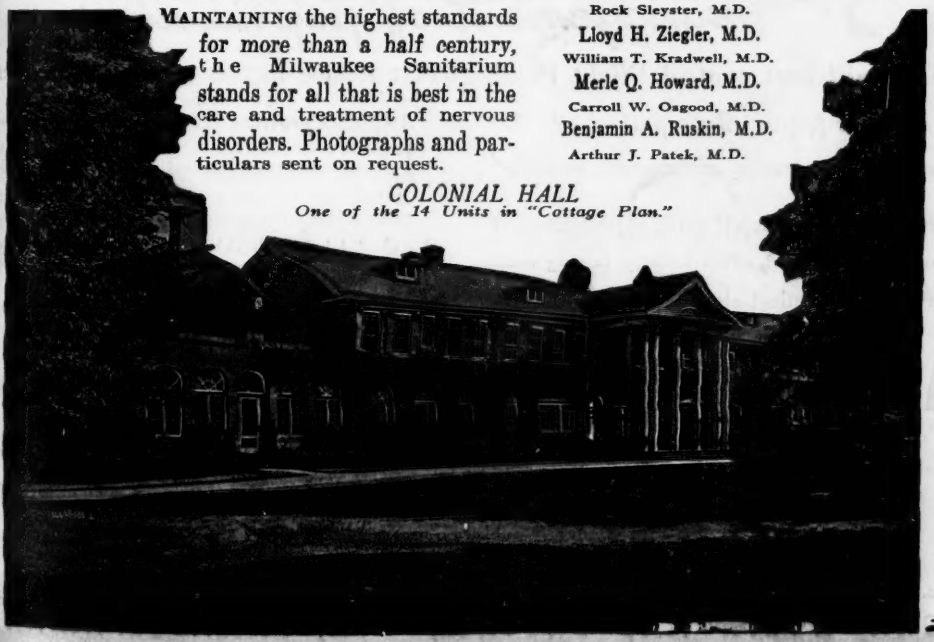

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The Illinois Medical Journal

October 1941

VOL. 80, NO. 4

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Editorials

AID TO DEPENDENT CHILDREN

During the last session of the Illinois Legislature, bills permitting the state to participate in the Social Security program of aid to dependent children were approved. Governor Green signed the bills so that they became effective on July 1st. Awards will be paid after October 1, 1941. Sixteen million dollars were appropriated for the biennium, 1941-1943, one half of which will be paid by the state and one half by the Federal Government in accordance with the Social Security Act.

The law will be administered by the State Department of Public Welfare through the County Departments of Public Welfare, which have been set up previously in each county of the state. Within the Department of Public Welfare the former Division of Old Age Assistance was changed to the Division of Public Assistance. It has been estimated that there are at least 70,000 needy children in Illinois who will receive benefits under this Act.

Approximately 17,000 children have received aid under the Mother's Pension Law, and 53,000 have been cared for through public funds, the total cost to the state under the new law will be approximately the same, while an additional four million dollars a year will be allotted by the Federal Government.

We are indebted to the Welfare Bulletin for the following information:

PROVISIONS OF THE LAW

The law provides that:

"A dependent child as the term is used in the Act means a needy child under the age of 16, or under the age of 18 if in regular attendance at school, who has been deprived of parental support or care by reason of the death, continued absence from the home, or physical or mental incapacity of the parent and who is living with his father, mother, grandfather, grandmother, brother, sister, stepfather, stepmother, stepbrother, stepsister, uncle, or aunt, in a place of residence maintained by one or more of such relatives in his or their own home.

"The child, to be eligible for aid must have resided in the state for one year immediately preceding the application for such aid; or have been born within the state within one year immediately preceding the application, and his mother must have resided in the state one year immediately before the birth of the child.

"Application for aid for a dependent child shall be made by the parent or relative with whom the child resides. The County Department of Public Welfare shall make an investigation and examination of the circumstances of the child, taking into consideration the suitability of the home in relation to the standards of care and health fixed by the laws of the state and by the rules and regulations of the Department of Public Welfare, including the social and educational opportunities of the child and the fostering and protection of its particular religious faith.

"The aid granted for a dependent child shall be determined in accordance with the rules and regulations of the Department of Public Welfare, taking into consideration the child's re-

quirements and the condition existing in the case, the income and resources available to him, from whatever source, and shall grant such aid to the child as shall be sufficient when added to the income and resources available to him, to provide him with a reasonable subsistence compatible with health and well-being.

"Names of persons receiving or applying for assistance and records concerning them shall be absolutely confidential except for purposes directly connected with the administration of the Act and shall not be divulged either directly or indirectly for any other purposes. No public official, agent, or representative in carrying out any of the provisions of the Act, shall take charge of any child over the objection of either the parent of the child or the person standing in the position of parent to the child, except by Court order."

The Mother's Pension law is repealed although the repeal does not take effect until July 1, 1942.

Under the Aid to Dependent Children program monthly payments will continue to be based on need, but the maximum will be the same over the entire state. The Federal Government will pay to the state an amount equal to one half of the total expenditure of the State up to $18.00 per month for one dependent child, and $12.00 per month for each of the other dependent children in the same home.

Under the Mother's Pension program payments have been subject to local tax levies and in some counties the taxable property has such a low assessment value that, even though the maximum levy for Mother's Pension is made, the amount subject to collection is inadequate to pay the county's share of the Mother's Pension costs. Under the A.D.C. program all counties will share equally, all must participate and there shall be uniform grants regardless of the amount of taxable property thus giving children in poorer counties equal advantages with those in more prosperous areas.

Physicians will have certain responsibilities in connection with the aid to Dependent Children program, as provisions should be made to give adequate medical care to these recipients. In the consideration of dependency, frequently examinations of the parents must be made before the decision can be reached.

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The Illinois State Medical Society has an Advisory Committee on Medical Care for Public Assistance Recipients which has met with officials of the State Division over a period of eight months, in an effort to develop a program which will be mutually satisfactory. County Medical Societies likewise, have named a similar committee to confer with the county Welfare Departments and assist them in caring for the many medical problems which will arise as a result of the operation of this program.

The State Division of Public Assistance and County Departments of Public Welfare likewise are responsible for the operation of the Old Age Assistance program, and a program for medical care for these recipients has also been developed through these repeated conferences. The program has been submitted to the county society committees, and arrangements have been made to conduct informative meetings in each Councilor District in Illinois, so that the local committee personnel will become more familiar with the provisions of the program, and likewise will know more about the duties they are expected to perform.

It is the opinion of the Council of the State Medical Society as well as of the Council Committee on Medical Care for Public Assistance Recipients that the program recommended for Old Age Assistance Recipients should be used likewise in the program of Aid to Dependent Children, and perhaps to the other public assistance groups to be considered later.

It is generally recognized in considering these programs that medicine is dealing with people of very low income, and that this fact should receive paramount consideration in the development of the programs for medical care.

MEDICAL COOPERATION IN THE DEFENSE PROGRAM

The medical profession throughout the country is cooperating in every way possible with the government in the vast program for National Defense. During the period from 1775 to the present time physicians have invariably responded to all calls from the government for service. Physicians have had an important role in all wars in which our country has participated.

We have been informed by historians that at the Battle of Bunker Hill in 1775, there were 36 physicians on duty and one of the most prominent colonial physicians, General Joseph Warren, was killed during this encounter. Another prominent physician during the Revolutionary War, Hugh Mercer, became a General. It is generally known that the first signer of the Declaration of Independence was Josiah Bartlett, physician and war surgeon.

Illinois physicians are cooperating in the present program in many ways. Hundreds have left their practice and families and are in army or naval service. We have in Illinois, at the present time, 360 local draft boards, and 40 medical advisory boards, and there are physicians devoting a considerable part of their time in making the necessary examination of selectees for each of these types of boards.

A recent check up gives the following information:

The total number of physicians in Illinois who have qualified as examiners for local boards, is 1705 and there are 557 appointed dentists.

For the 40 Medical Advisory Boards in Illinois, there are 526 physicians and 94 dentists who have qualified up to September 1, 1941.

In addition to these appointments, there is one physician on each of the Appeal Boards, the third type of Boards in the Selective Service set up within the several states.

This makes a total of approximately 2245 physicians and 620 dentists who are acting in some capacity in Illinois, for the examination and classification of men called before the local, medical advisory and appeal boards.

Surveys have been made to determine the needs for medical care of the civilian population as well as workers in essential industries, so that both groups will receive adequate medical care in the present emergency.

Another important matter which will require a considerable amount of attention is the return to normalcy following the demobilization period which everyone hopes will be forthcoming within a reasonable period of time.

Medicine once more has received a definite responsibility in this unusual emergency, and every effort has been made to give the best possible service to the Government, as has been the case in all previous emergencies since the birth of this Nation.

Correspondence

BRITISH PLASTIC SURGEON TO ADDRESS CHICAGO MEETING OF SPECIALISTS

Sir Harold Delf Gillies, one of Great Britain's leading plastic surgeons, who is now in charge of special units for the repair of facial injuries in the London area, is to be a guest of the American Academy of Ophthalmology and Otolaryngology, national organization of eye, ear, nose and throat specialists, when it meets in Chicago at the Palmer House, October 19-23.

Sir Harold will address a special defense program presented by the Academy on Monday evening October 20. The other speakers will be Dr. Irvin Abell, Louisville, Ky., a former president of the American Medical Association and now chairman of the association's committee on medical preparedness as well as chairman of the health and medical committee of the Federal Security Agency; Col. Louis H. Bauer, Hempstead, N. Y., an authority on aviation medicine and now a member of the New York state medical society's committee on medical preparedness, and Dr. Burt R. Shurly, Detroit, a former president of the academy and chairman of its special committee on national defense. Dr. Samuel J. Kopetzky, chief of the medical division of the selective service system in New York City, will be the narrator for a series of motion pictures on the medical services of the Army and Navy.

Sir Harold, a native of New Zealand, is plastic surgeon to the great St. Bartholomew's Hospital in London and has been on the staffs of other hospitals at various times. At present he is consultant adviser to the ministry of health and is in charge of the three plastic surgery units in London. It was recently reported to the American Medical Association that Sir Harold's house had been bombed and that his eldest son was taken prisoner following a forced landing of his plane.

Sir Harold is an honorary member of the American College of Surgeons and of the American Dental Association. In 1935 he delivered the Mayo Lecture at Northwestern University Medical School, Chicago.

ANNOUNCEMENT OF VAN METER PRIZE AWARD

The American Association for the Study of Goiter again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held at Atlanta, Georgia, June 1st, 2nd, and 3rd, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten, double spaced copy sent to the Corresponding Secretary, Dr. T. C. Davison, 478 Peachtree Street, Atlanta, Georgia, not later than April 1st.

The Committee, who will review the manuscripts, is composed of men well qualified to judge the merits of the competing essays. Dr. Asher Chapman of Rochester, Minnesota, received the Award for the year 1941 in recognition of his essay entitled "The Relationship of the Thyroid and the Pituitary Glands to Iodine Metabolism and Extrathyroid Iodine Metabolism."

A place will be reserved on the program of the annual meeting for presentation of the Prize Award Essay by the author if it is possible for

him to attend. The essay will be published in the annual Proceedings of the Association. This will not prevent its further publication, however, in any journal selected by the author.

SIMPLIFIED PRACTICE RECOMMENDATION R106-41, HOSPITAL PLUMBING FIXTURES, NOW IN PRINT

Printed copies of Simplified Practice Recommendation R106-41, Hospital Plumbing Fixtures, are now available, according to an announcement of the Division of Simplified Practice, National Bureau of Standards.

In May, 1929, a general conference of hospital administrators, representatives of plumbing fixture manufacturers, and others concerned, approved a simplified schedule of standard stock plumbing fixtures for hospitals.

The Standing Committee in charge of this recommendation drafted a proposed revision in 1939, which was referred to the manufacturers joint committee on standards and specifications for further study. At a meeting of this committee in May, 1940, a final revision was drafted and approved. This proposed revision was then approved by the industry for promulgation by the U. S. Department of Commerce, effective from July 1, 1941. In addition to the list of standard fixtures, the recommendation also includes reference to grading rules, nomenclature, tolerances, methods of test, installation, etc.

Copies of R106-41 may be obtained from the Superintendent of Documents, Government Printing Office, Washington, D. C., for 5 cents each. A discount of 25 percent on orders for 100 or more copies is available to those desiring a supply of this publication for business purposes.

Roland R. Cross, M.D., Director Illinois State Department of Public Health recently released the following information.

TWO TYPES OF BIRTH VERIFICATION FORMS

With the adoption of the new amendment to the Vital Statistics Act in Illinois there are now two different types of birth verification forms available according to State law. Called a "certification of birth registration," the new abbreviated form *attests* to the fact that the birth in question has been duly recorded, and *establishes* the name, sex, date, and place of birth of the person to whom it relates. This information provides adequate legal proof of age and of citizenship, without revealing detailed personal data as to parentage, legitimacy, etc.

The older type of form is a certified copy of the original certificate of birth, and has been in use since 1916. This contains all the information given in the short new form plus a number of personal items not usually of interest to the agencies and organizations which demand to see birth verifications.

Either type of form is available for the statutory fee of fifty cents. If the birth was reported before 1916 the facts (on either form) should be available from the clerk of the county in which the birth occurred. Verifications of Illinois births registered since 1916 may be obtained from the local registrar, the county clerk, or the Illinois Department of Public Health.

TO LECTURE ON PSYCHOLOGICAL EFFECTS OF WARFARE

One of England's foremost specialists in psychiatry and neurology has been granted a leave of absence from the RAF by the British government to report to the American medical profession his first-hand observations on the psychological effects of "blitz" warfare on armed forces and civilian population.

Dr. R. D. Gillespie, who is now chief psychiatrist for the British Royal Air Force, is coming to this country at the request of the Salmon Committee on Psychiatry and Mental Hygiene of the New York Academy of Medicine. He will deliver the Salmon Memorial Lecture in the New York Academy building Nov. 17, 18 and 19, speaking on "Psychoneuroses from the Standpoint of War Experience."

The New York lectures will be followed by addresses before the Chicago Neurological Society, the Chicago Institute of Medicine and the Illinois Psychiatric Society in Chicago. Dates for these lectures as well as lectures in Toronto, Ontario and San Francisco, California, will be announced later.

Dr. Gillespie's observations made under actual war conditions are expected to be of inestimable value to American psychiatrists in formulating plans for maintaining civilian morale in wartime. His lectures will also give his hearers new information concerning the pressing psychiatric problem of today — the psychoneuroses.

Dr. C. Charles Burlingame, chairman of the Salmon Committee which each year chooses an outstanding specialist in psychiatry, neurology or mental hygiene for the Salmon Memorial Lecture, has issued a general invitation on behalf of the Committee to members of the medical profession and their friends to attend. Dr. Gillespie will be the 9th lecturer who has been selected from the top-ranking psychiatrists and neurologists throughout the world who have made the greatest contribution to their field during the preceding year. Selection for delivering ing the preceding year. Selection for delivering tion for the Pulitzer Prize in letters.

Dr. Gillespie is the author of two medical volumes "Disorders in Sleep" and "Textbook of Psychiatry," a reference work in collaboration with Dr. D. K. Henderson of Edinburgh, Scotland, who is a former Salmon Lecturer. Before he undertook his wartime duties as chief psychiatrist for the RAF, Dr. Gillespie was the lecturer in Psychological Medicine at Guys Hospital and Medical School in London. He was also the lecturer in Psychopathology at the University of Aberdeen. He received his medical degree from the University of Glasgow and his doctorate in psychological medicine from the University of London. He is a member of the Royal College of Physicians.

Among Dr. Gillespie's many important findings concerning the psychological effects of modern warfare on civilians and combatants is the fact that chronically neurotic people and those suffering from mild depressions have become chronically heroic and self-sacrificing under the stress of bombing raids. Also, he has noted that women stand up as well if not possibly better than men under civilian bombings.

Permission for Dr. Gillespie to come to the United States for the specific purpose of delivering the Salmon Lectures was secured, after several months' negotiations from Sir Harold Whittingham, Director General of the Medical Services of the Air Ministry in London. Joining the Salmon Committee on Psychiatry and Mental Hygiene in its appeal to the British Government, were the following doctors:

Dr. Winfred Overholser of Washington, D. C., Chairman of the Psychiatric Committee of the National Research Council; Dr. James K. Hall of Richmond, Va., President of the American Psychiatric Association; Dr. C. Macfie Campbell of Boston, Mass., President of the American Board of Psychiatry and Neurology and Dr. Louis J. Pollock of Chicago, Ill., president of the American Neurological Society. These doctors represent the leading groups of Neurologists and Psychiatrists in America.

To the Editor:—

The following is the list of officers and committees for the Woman's Auxiliary which we would like published in the Illinois Medical Journal.

Sincerely,

Bernice L. Stigman

State Press & Publicity Chairman

Woman's Auxiliary to the Illinois Medical Society

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OUR AGING POPULATION

In the early days of this country there was a large proportion of children and young adults, with a correspondingly small proportion of older persons. It will not be long now, trends being what they are, when this state of affairs will be reversed.

Three factors are operating to produce the latter result — declining birth rate, curtailed immigration, and improved mortality in childhood and early adult life.

Dublin foresees some interesting (and disquieting) consequences. While normally conservative and tending to uphold sound political and social institutions, the elderly may be manipulated unscrupulously and demand grants ruinous economically and financially, including the gratification of special medical desires and needs. New types of hospitals for mental and physical ailments, particularly in southern areas, with a vast army of government-employed physicians, will be some of the effects of medicine.

Small apartments will be the rule. Per capita food consumption will tend to decline — a blow to agriculture. The payment of old age benefits under social security will be alarmingly accelerated.

Future federal budgets will have to take the aging population constantly into account, along with new pensions for war service.

The new pattern of life will call for statesmanship of a high order, if disaster is to be averted and advantage taken of whatever is promising in the new order of things.

But the conclusion can not be escaped that we are imposing upon our children an obligation to care for a huge proportion of old people, along with the debts now being incurred for other reasons.

The race suicide fanatics may yet face a fearful reckoning. Dublin loks for a minimum of about 13 births per thousand of population by 1980. He so reads the "wave of the future."—*Medical Times.*

THIS TRIFLE, WAR

Whatever its drawbacks, total war is not open to the same eugenic objections as the old-fashioned kind. It has been said of wars in the past that they destroy the cream of the young manhood of a generation; and that the best germ-plasm is thus irrevocably lost, to the lasting detriment of the race. To see men in terms of germ-plasm savours, perhaps, of the biologist's fancy that the hen is merely a device for producing more eggs; but the argument is disturbing for all that. To know that this dysgenic effect is reduced, because the risk is more evenly distributed, is a minor consolation of this war. Raymond Pearl, however, held that no war had so far occurred in the nineteenth or twentieth century which had had any measurable genetic effect on the nations involved. This seems incredible until his figures are studied. He pointed out that wars, nowadays, last only a short time in comparison with the span of human life; and more fighters return than are killed. They return in the prime of life, capable even if maimed of founding healthy families; and most of them do. In the last war those killed formed a startlingly small fraction of the total population. He estimated that the loss to the American race was only 0.055% of the 1910 population, or 0.048% of the 1920 population, and thought it unlikely that any geneticist would regard five one-hundredths of 1% as a significant loss from the total gene pool of the population. The comparable figure for France was, of course, much higher, lying, he calculated, somewhere between 1.4 and 3.4% of the population; yet even so it could have had no measurable effect on the quality of the French race. These figures are worth remembering at the present time, when pressure of war spoils the sense of proportion. Nor is it merely on genetic grounds that they are cheering; they remind that, at worst, wars are minor interruptions of our evolution and that we have a good chance of growing out of them.—*The Lancet, June 7, 1941.*

Medical Economics

Edited by R. K. Packard, M.D., Chairman of the Committee on Medical Economics of the Illinois State Medical Society, 826 East 61st Street, Chicago, Illinois.

Reading, today, the current literature on our political, social economics, labor management, production, agriculture and distribution problems along with the problems of taxes, and the function of government one arrives at a state of confusion. Perhaps this confusion is the result of distortion of facts, lack of facts and failure to present in a logical way the pros and cons of their arguments. Attempting to find myself and to clear up part of my confusion, I read again Adam Smith on "The Wealth of Nations." For better understanding I read it again. Published in 1776 it belongs to the horse and buggy days. It has lived through these years as perhaps the best contribution on political economy. I would suggest that doctors interested in economics read it, and that legislatures, executives, social leaders and labor leaders also read it. There are certain fundamentals that must be preserved if democracy is to survive. We must work, we must be frugal, we must be honest, we must be tolerant, we must accept individual responsibility, we must accept our responsibility to others according to our position and station in life. We must not assume that the function of government is to support us, but rather that our function is to support the government that is essential to society. We have reached the highest point of universal education, in this country, of any country in the world and if education is of value we should need less and less of planned economy and have more and more of individual planning of our own economy. It would seem to me that those responsible for our educational policies should strive for that goal rather than for a planned economy.

Returning to our own subject of medical economics, I sometimes wonder if our conception of this subject is clear. Some doctors think of the subject only as it provides for and protects their financial gain. Outside of the profession some think of it only as it relates to the public with no consideration of the financial return to the doctor. We cannot have a producer and a consumer at great odds without harm to both. If we have a poor product, we loose our market. If we have a good product the consumer and producer both profit. Competition tends to produce a better product which benefits both producer and consumer. Medical care is a product. The consumer is best served by the best product. Competition and quality usually adjust price levels. Can we increase the consumption of medical care, retain its present high quality and leave a profit to the producer, commensurate with his investment, his hours of labor, his great responsibility, his depreciation of plant equipment, which is essentially himself? As stated in previous articles experiments are being carried on to increase the consumption of medical care at a lower cost to the individual, yet preserving the quality of the service and even improving it.

We hear no great cry for cheaper automobiles. Our demand is for quality and safety. They cost more than a horse and buggy used to. They give us better transportation. Manufacturers who have been able to produce quality cars and improve them every year have survived. Quality transportation, in spite of the original investment, has proven profitable to producer and consumer. Medical care in the automobile and air plane era costs more than the medical care of the horse and buggy era. Quality has improved. The health of the nation is better. The consumer has profited. Medical economics does not look to producer profits as its legitimate goal. It looks to quality production and consumer value in health. While the cost of medical care has increased; we must pause and take notice of the millions of dollars that have been saved and

more important the millions of lives that have been saved by the reduction in infant and maternal mortality and morbidity, by the nearly complete eradication of typhoid fever, malaria, diphtheria, scarlet fever, by the reduction in the mortality in pneumonia by fifty percent, by the constant reduction in tuberculosis and the cure of many cases now that at one time were considered hopeless, by the prevention of deformities resulting from infantile paralysis by the skill and knowledge of orthopedic surgeons. Many other examples could be cited. The medical profession is constantly endeavoring to reduce the incidence of disease. It is a quality product which we hope to maintain for producer and consumer benefit.

Dr. R. K. Packard, Chairman
Medical Economics Committee

NURSING EDUCATION VS. NURSING CARE

From a discussion at the midwinter session of the Council of our Association, it is clear that the problem of nursing care is constantly growing in seriousness and that unless remedial measures are promptly instituted there is danger of a complete breakdown of this indispensable service to the sick. Hospitals throughout the state are threatened with serious handicaps in their nursing care. The problem is particularly acute for smaller institutions in the outlying districts.

The present dilemma may be traced to a number of factors. The public health activities, the Red Cross, the Visiting Nurse Associations, the school health services, all have already absorbed, and are continuing to draw, goodly numbers of graduates. The salaries, the hours, and the nature of service offered by these positions are obviously more attractive than are the duties connected with bedside routine in a home or in a hospital.

The greatest contributing factor, however, must be sought in deeper territory. The history of nursing education in this country runs closely parallel to that of medical education with this exception. A generation ago there were 170 medical colleges in the United States. As to training schools, well, there were almost as many as there were hospitals. In Nebraska too, until the reform began it was unthinkable for a respectable hospital to deprive itself of a nurses' training school. This statement is not made in a spirit of sarcasm. Indeed, many graduates from these now extinct training schools are today rendering excellent bedside care to the people of our commonwealth.

With the change in trends of education in nursing, the smaller schools have disappeared. Now only the large and well endowed hospitals can keep up with the rigid requirements and the academic equipment necessary for an approved training school. The effort to improve the standards of any profession deserves praise, and as physicians we express our enthusiasm over the attempt. In fact, we have helped in no small measure to expand the curriculum.

"The recently graduated nurse is more interested in an executive or administrative job than in actual nursing." This statement was made in full sincerity by a member of the Council, who for many years has operated a well equipped but small hospital in the state. The sentiment seemed to be general and practically unanimous that the modern R.N. considers herself too well trained to administer "hypos" and enemas.

It is not within the scope of this editorial to suggest a solution to the problem. That the situation is urgent the leaders in nursing education undoubtedly appreciate. It is ardently hoped that our committees appointed to study the dilemma will bring in some tangible information that may promptly be utilized as a basis for betterment of nursing care in Nebraska.—Editorial from *Nebraska State Medical Journal,* March, 1941.

NAVAL MEDICAL CORPS

The Medical Corps of the Navy offers an interesting career. Just as in private life, much depends on the individual. In normal times, a physician who has completed his internship, has graduated from a grade A school and has passed successfully mental, moral and physical examinations is first sent to one of the base hospitals of the Navy or to the medical school in Washington for what is termed "indoctrination." This does not mean strictly professional training. It means that the young physician is taught the various naval forms and procedures, naval etiquette. At first, it all seems rather complicated, but the young physician soon begins to feel that he has arrived. He begins to feel that he has mastered the so-called red tape of the service. It is necessary for officers of the Navy to be well-trained, not only professionally but also from a naval standpoint. Each officer must know the laws and customs of the naval service.

At the completion of his training, the young doctor begins his naval career. Usually he goes to sea or to some tropical station. Many of these are delightful spots. I recently spent more than two years as chief surgeon at the naval hospital in Pearl Harbor, Hawaii, and I must admit that those years were some of the happiest of my life.

After the physician's first cruise or tour in the tropics, which usually lasts approximately two years, he is privileged to choose a course in medicine or surgery in which he is most interested. On completion of this course, he is assigned as assistant to one of the senior officers. Throughout his career, he may request these special courses in order that he may keep abreast of the latest developments.—*G. A. Eckert, Commander (MC) USN, in Staff Meetings of the Mayo Clinic, April 30, 1941.*

Original Articles

THE DIAGNOSIS OF CARCINOMA OF THE ESOPHAGUS

JAMES B. COSTEN, M.D.
AND
WM. T. K. BRYAN, M.D.
ST. LOUIS

Diagnosis of carcinoma of the esophagus is rarely made early in the course of this not too infrequent disease. This disease, as we all know, carries an extremely bad prognosis. The few cases that have been cured, as in cancer elsewhere in the body, had early lesions. Improvement in this situation can be expected only by more intensive search for early cases. Hence, a study has been made of six cases whose findings were pertinent in the differential diagnosis, and of the 106 cases admitted to Barnes Hospital and the Barnard Free Skin & Cancer Hospital during the past decade, in an effort to correlate and emphasize the symptoms which might stimulate interest in an earlier diagnosis of this disease. Enormous numbers of reports fill the literature (Bird); however, the gravity of this problem justifies continued discussion.

Carcinoma of the esophagus is not only fascinating as a study, but an important problem, as it is as yet neither successfully diagnosed in the early stage nor treated with more than rare success. Its early diagnosis will be obtained only at the expense of routine complete examinations of all unexplained cases of dysphagia, pain in the chest, hoarseness, or vomiting. The treatment is necessarily strenuous, whether so-called palliative or curative, by radiation or surgery, so that the typical wasted victim soon succumbs.

Esophageal cancer has a fairly typical course with the primary symptom unfortunately common to other esophageal diseases, namely, some disturbance with swallowing. In this series of cases from Barnes and Barnard Hospitals 75% gave it as an initial symptom. This is in line with other large series reports. However, there are approximately 60% of all cases of dysphagia who do not have cancer, as shown by Macmillan[1], in his analysis of 1600 cases.

It is not too extravagant to suggest that carcinoma of the esophagus should be borne in mind in any case presenting to the doctor the symptoms of belching with regurgitation, accumulation of excessive saliva, sensation of lump in the throat, with or without mild choking sensations, substernal distress of any kind, or cough without fever, and upper respiratory infection. Hoarseness is purely a laryngeal symptom, but may be due to a recurrent paralysis from aneurysm or compression of an esophageal growth. A case with one or more of these symptoms might have any of the following conditions:

1. Simple hyperacidity of the stomach, the convenient and too easily acceptable blanket to cover a multitude of gastric disturbances.
2. Cardiac lesions, which when simple, respond to reassurance, a sedative and a rest period; when serious recognizable by the ordinary means of cardiac diagnosis.
3. Esophagitis, a condition occurring far more commonly than ever suspected; is characterized by substernal burning pain increased by swallowing solid food, occasional clearing of blood from the throat, and here again dysphagia; it is difficult to differentiate from lesions of the stomach, gall-bladder and intestines, and can be diagnosed only with certainty with the esophagoscope; it has its origin in vomiting, trauma from the passing of tubes,

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From the Departments of Otolaryngology, Washington Univ. School of Medicine, & The Oscar Johnson Institute; & the Barnard Free Skin & Cancer Hospital, St. Louis, Mo.

and in foci of infection about the sinuses and throat.

4. Achalasia of the esophagus, "cardiospasm" to most observers, is actually a diffuse dilation of the esophagus and failure of the diaphragmatic pinchcock to open, according to Jackson[2]; but here again characterized by slowly increasing dysphagia at times and regurgitation.

5. The hysterical dysphagia of Plummer and Vinson[3], is typified by gradual onset of difficulty in swallowing solid foods, glossitis, anemia, and enlargement of the spheen.

6. Globus Hystericus, a term which has been discarded by many observers, actually is seen in persons sustaining psychic shock, and in young girls at adolescence; the lump in this patient's throat is more imaginary than real, swallowing is not actually impeded, and the patient is managed by neurological routine rather than diagnostic procedures, the simplest of which makes the problem more difficult.

7. Diverticulum of the esophagus, a pulsion phenomenon caused by overaction of the criopharyngeal muscle when at that level, or by inflammation of the muscular esophageal wall when lower down, is a disease of middle age, rarely occurring before forty; it is recognized by gurgling noises when swallowing food and the recovery of excess secretions in the mouth during sleep.

Carcinoma of the esophagus, rare under forty years of age is five times more frequent in the male, more commonly recognized in recent decades as the leading basis for esophageal obstruction, extremely malignant, and so far, an entirely uncontrolled disease. Symptoms, when present, are typical.

1. *Dysphagia:* Subjective symptoms, of the mildest grade, form the early basis of diagnosis of carcinoma of the esophagus, and the first is disturbance in normal swallowing, long before actual difficulty is noted. The patient is reluctant to see the doctor for such a complaint, but notes its persistance. A stage is reached when food "sticks in the throat," has to be masticated carefully or is painful when taken in average amounts, and is regurgitated a few seconds after ingestion, even liquids. Choking, when it occurs at this stage may be due either to the constriction or to spilling over of liquids into the trachea. This would indicate almost complete closure of the lumen of the esophagus. It is likely that "vomiting" described in this connection is really regurgitation. It has been called "esophageal pseudo-vomiting." Actual difficulty in swallowing food is uncommon in neurotic patients.

Esophageal dysfunction was already present as a definite symptom in 100 of the 106 cases in this series by the time of admission to hospital, of the remaining six three had been vomiting and a diagnosis made by Roentgen study with barium swallow. Some difficulty in swallowing was given as the initial symptom in 82 cases. Pain was initial in only 11 cases, hoarseness in 5, cough, vomiting or choking each in 2 cases.

2. *Pain:* Actual painful sensations are contrary to the rule until closure of the esophageal lumen has progressed to an obstructive extent, but certain cases have a vague discomfort or fullness beneath the upper or lower part of the sternum. "Heartburn," an epigastric pain when food is taken, may be the first sign of ulceration in a malignant lesion at the cardia. A sticking sensation in the throat on swallowing may denote hypertrophy or inflammation of the lingual tonsils. Complaint of a lump or tightness in the throat is often associated with hypothyroidism. Mild symptoms in esophageal cancer are conspicuous by their scarcity. This is taken by Vinson[4] to indicate that there are no early symptoms and that from the very beginning of symptoms the lesion has already progressed extensively.

The patient may note that food seems to stop at a certain level with a sense of oppression beneath the sternum in the same plane. It has been stated, (Abel[5]), that the level of this sensation may have no relation to level of the lesion in the esophagus. However, recent experiments notably by Jones[6], indicate that levels actually are correlated. After introducing a distensible balloon attached to a fine rubber tube to a definite level of the esophagus and stretching it so that "In the upper third the localization was for the most part in the midline at the episternal notch or directly under the first portion of the manubrium . . . when the balloon was moved to the level of the mid-esophagus each of twenty-five subjects referred any sensation to the midline. In most instances the localization was sharply defined. Eighteen localized it almost

over the balloon, deep under the middle of the sternum; . . . . . with distention of the balloon in the lower third of the esophagus at a point just above the cardiac end of the stomach, all but four patients experienced a well-localized sensation of discomfort in the midline under the xiphoid." He noted that reference of the sensations caused by distention of the esophagus was fairly constant, but that the character of the discomfort varied a great deal.

In this group of cases the sensations were described as follows in order of frequency; fullness, pressure, burning, uncomfortable sense of constriction, sharp pain, prickling sensation, acid taste, aching and numbness over the chest. In 42 cases the location of pain was described and the level of the lesion was noted. It was found that there was a rough but definite correlation of levels of lesion and pain — pain in or above the sternum came from lesions in the upper third eight times, in the middle one time and in the lower once. Two cases had pain referred to the ear with lesions at the cricopharyngeus. Lesions in the middle and lower thirds had substernal pain in about equal numbers, that is, thirteen middle, twelve lower and two upper.

3. *Hoarseness and Cough:* Hoarseness, which is present only occasionally, is due to recurrent paralysis when the tumor involves a recurrent laryngeal nerve by extension of carcinoma of the cervical esophagus. Cough is more common and is the result of spilling of excess mucus into the larynx, or spilling over of fluids when swallowing is attempted.

Case 1. J.F., a 72 year old white man, complained of increasing dysphagia with regurgitation for five months previous to his admission to hospital on May 4th, 1937. He had never brought up blood, and there was no loss of weight. Besides a moderately advanced arteriosclerosis, he had a bilateral inguinal hernia. He also had a marked kyphosis and left scoliosis with very bent hunch-back effect, from an injury to the spine sustained twenty years before.

Barium x-ray revealed that "the lower third of the esophageal tract did not indicate complete obstruction but the irregular defect presents suggestive wall infiltrations, probably malignant. Fluroscopically for a period of five minutes the barium meal showed decreased motility or partial obstruction." One week later "re-examination of the esophagus after administration of antispasmodic medication does not show any relaxation of the strictured area. The present findings point more definitely towards malignancy."

On August 20, 1937, gastrostomy was done by Drs. Sherwin and Martin because of complete obstruction, and urgent nutritional need. He was carried along on tube feedings at home, and did well on digitalis routine.

Esophagoscopy was considered safe on the basis of his improved heart condition, and was done on March 5, 1940. "At 25 cm distance from the incisor gums the lumen narrowed, to a stricture-like opening similar to lye scarring. (Fig. 1) Although very small, it was easily dilated with stiff catheters, which passed to the stomach. No evidence of malignancy was seen." No reaction followed the dilation, and he began at once to swallow liquids well. In spite of advice, he did not return for further dilatations.

Fig. 1: 2-28-40. J.F., "Barium x-ray shows the esophagus well outlined in lateral view. There is a localized area of narrowing in the lower third of the esophagus. The proximal portion of the esophagus is slightly dilated. The portion of the esophagus distal to the narrowing is normal in caliber. A lateral view of the thoracic vertebrae reveals an old compressed fracture of the tenth vertebra, this being just opposite the narrowing in the esophagus. It is quite logical to assume that the narrowing seen in the esophagus is the result of some fibrosis and periesophageal adhesions directly related to the trauma sustained twenty years ago." Dr. E. C. Ernst. (This was confirmed at autopsy after the patient's death at St. Louis City Hospital, six months later.)

Case 2. R.S., a man aged 72 years, was an employee of the hospital. He was treated during April and May 1940 for an almost complete exfoliative dermatitis. He was first seen by the Ear, Nose and Throat Service on August 2, 1940, with the complaint of hoarseness of three weeks duration. This came on suddenly and did not improve. His voice became falsetto. There was no previous trouble with the voice on

other complaints. Examination revealed paralysis of the left vocal cord and fixation of the arytenoid. X-ray of the chest was negative. Accordingly on September 9th, a Lynch suspension laryngeal examination was done. The only finding was slight swelling of the left arytenoid. He was re-admitted September 4th, because of the skin condition. Chest x-ray was repeated for possible mediastinal mass but it showed only thickened pleura. He began to lose weight and have difficulty in swallowing in October. On December 22nd a barium swallow was done. It was reported that some of the barium spilled over into the trachea and this was interpreted as due to the paralyzed vocal cord. February 4th, he was re-admitted to the hospital much weaker, unable to swallow fluids freely, and having lost 35 pounds of weight. X-ray was advised with special attention to the mediastinum. A barium swallow showed a filling defect in the upper half of the esophagus and there was a spill-over into the trachea. Esophagoscopy was done on the 8th; revealing a hard annular mass at the cricopharyngus 12 cm long. It was dilated and biopsied. The biopsy showed carcinoma, squamous cell. He was given x-ray palliatively. He became weaker and died March 13, 1941.

Case 3. G.H. This was a 65 year old waiter who gave the history when admitted to Barnard Free Skin & Cancer Hospital on January 7, 1941, of dysphagia for 2½ months and extreme hoarseness for three weeks. Besides general emaciation he showed enlarged firm cervical glands above the right clavicle. Laryngeal examination revealed fixation and enlargement of the arytenoids, giving the impression of a lesion in the crico-pharynx. (Fig 2a-2b) Esophagoscopic examination was limited by an obstructing mass filling the crico-pharyngeal space, arising from the anterior surface of the esophagus. This tumor appeared to be primary in the esophagus and biopsy and reported as squamous cell carcinoma.

Following esophagoscopy respiratory difficulty increased and tracheotomy was necessary the following day. He ran a fever course incidental to a mild bronchopneumonia for two weeks, and was considered improved, enough for gastrostomy on January 21, 1941. Just before operation and before any anesthesia, the patient collapsed, developing increased reflexes over the right side. He was diagnosed as cerebral hemorrhage but ran a septic temperature beginning that day. Both from physical signs and x-ray study of chest this was determined to be an aspiration pneumonia, with pleurisy. The condition improved on sulfathiazol medication. Without change in symptoms the patient expired suddenly January 25, 1941.

Anatomical diagnosis at autopsy gave the primary site of the tumor in the larynx, with extension to the trachea, esophagus, and right lobe of the thyroid. Multiple abscesses of lower lobe of right lung and pleurisy were found.

Case 4. C.T., an unemployed white man age 66 was admitted to the hospital on November 7, 1940. He gave a history of prolonged malnutrition and neglect, vague in detail, except that two years before he had had a spell of diarrhea, occasionally bloody. He then

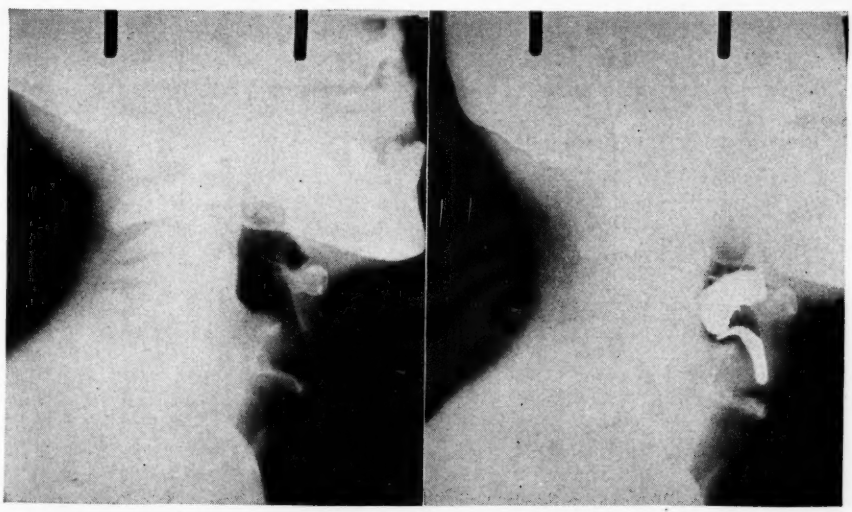


Figure 2a-2b: 12-31-40. G. H., "The lateral projection of the cervical or laryngeal region reveals an irregular mass arising from the superior portion of the larynx, posteriorly, and appears to be pushing forward. This can be noted by the displacement of the air column. (Fig. 2a) The epiglottis and hyoid bone are normally outlined. This displacement is more obvious after the administration of a small amount of barium. A complete obstruction of the upper portion of the oesophagus can be seen. (Fig. 2b) The mass is pushing forward against the upper portion of the thyroid cartilage. A small amount of barium can be seen in the ventricle. The A-P view reveals the mass to be arising from the left side, possibly the left pyriform fossa, because this area fails to fill with barium." (Dr. E. C. Ernst).

had increasing constipation, burning in the epigastrium with flatus, and gradual loss of weight. Burning and difficulty in swallowing came on gradually. "For two months there has been continuous pain and aching in the epigastrium, cough and rapid loss of appetite. For the past month has had pain in the chest, sore throat, and hoarseness. At present he has a paroxysm of coughing when he tries to swallow either solids or liquids. Sometimes there is a lancinating pain in the lower left chest." Examination revealed paralysis of the right vocal cord, weakness of the left, diminished gag reflex, and both pyriform sinuses full of saliva. The left posterior cervical glands were palpable. Heart sounds were soft, lungs clear, the blood pressure 160-90. The liver was palpable three fingers below the costal margin, smooth, and not tender. Laboratory examination showed urine to be alkaline, with specific gravity of 1.014. The blood count was r.b.c. 4.15 million, Hemaglobin 90%. He was esophagoscoped November 14 and 16. The scope passed easily to the cricopharyngeus where a hard granular non-ulcerated wall was encountered into which a thread passed but no lumen could be found.

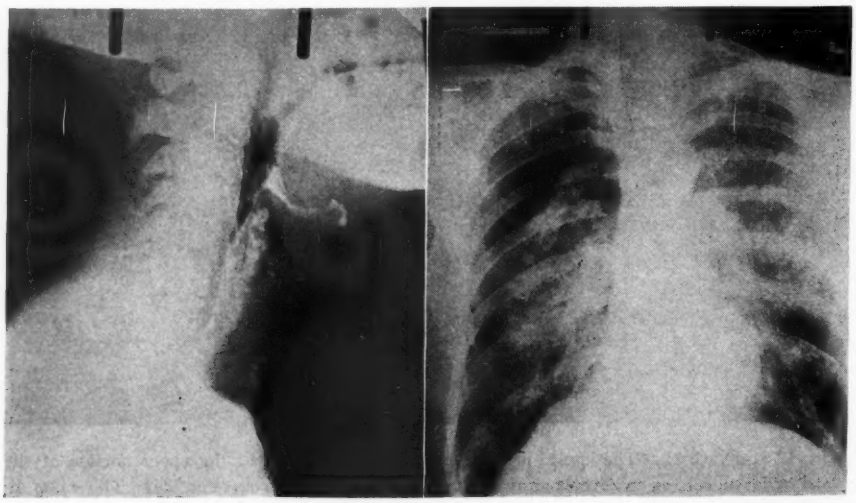
He was referred accordingly to Dr. Womack, who performed a gastrostomy on November 19, 1940. Biopsy of a skin nodule removed from the abdomen at that time showed metastatic squamous cell carcinoma. His nutrition failed to improve, and he died one month later.

Autopsy showed "an annular constriction of the esophagus at its origin from the hypopharynx, of firm white tumor tissue. The lumen is constricted to a narrow slit. There is superficial ulceration of the mucosa. The tumor extends forward into the wall of the trachea at a level 3 cm below the larynx, but without ulceration. There are metastatic nodules in the wall of the esophagus, in the adjacent lymph nodes in the thyroid gland and over the pleural surfaces, both visceral and parietal, extending to the domes of the diaphraghm."

Case 5. Wm. E. H., 54 years old sheet metal worker was admitted on February 18, 1941, to Barnard Free Skin and Cancer Hospital with the complaint of soreness in his throat, increasingly painful for one month. The condition began eleven months before, with steady loss of weight (30 lbs.), hoarseness and total inability to swallow recently Examination by his local physician produced no findings and he suspected cancer of the esophagus.

Laryngoscopic study on admission showed the left false and true cords fused in a large mass, in the center of which was a larger irregular ulcer, necrotic at its center, its edges raised. The left arytenoid was enlarged with a pale swelling. All laryngeal mucosa was pale and the pharyngeal mucosa dark red with heavy granulations. Biopsy taken from the edge of the ulcer showed no evidence of malignancy, and no evidence of microscopic tubercle formations. There were, however, large numbers of giant cells, and foci of necrosis in the inflammatory exudate. A tentative diagnosis of tuberculosis was made.

He was given a poor prognosis and referred for routine tuberculosis care.



3a 3b

Barium x-ray studies of the esophagus showed the lumen normal and the opaque material passed well. (Fig. 3a) A plate of the chest showed far advanced bilateral pulmonary tuberculosis with pneumo-thorax of the right upper lung, and collapse of 25% of its total volume. (Fig. 3b)

Case 6. K.M., a 56 year old white male, was admitted to Barnes Hospital on September 10, 1940, complaining of difficulty in swallowing for five months. Barium visualization showed tumor growth closing almost the entire middle third of the esophagus. (Fig. 4) Esophagoscopy for biopsy was done on admission, removing tissue from a spot at 26 cm level. This showed squamous cell carcinoma, of high malignancy. Dilatation as limited, and complete obstruction occurred within two days afterward. Janeway Gastrostomy was done by Dr. Womach on September 13, 1940, with very little reaction.

On September 21, 1940, 75 mgm of radium was applied within the esophagus, using six needles of 12.5 mgm each, placed in series within a rubber catheter. The catheter was passed after fixation to swallowed black silk. The total radiation was 1275 mgm hours, the treatment lasting 17 hours.

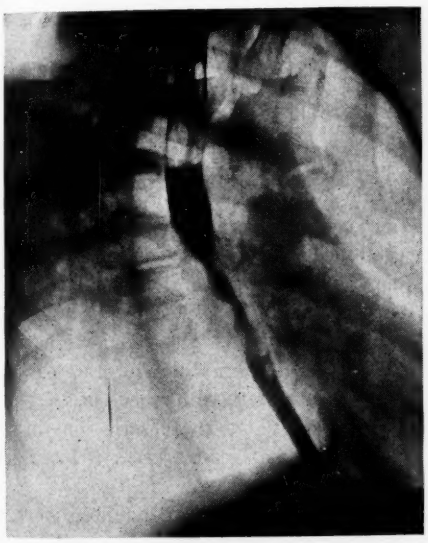


Figure 4. 9-10-40. K.M., "Special oesophagus examination shows a constricting, infiltrating lesion several centimeters long at the level of the bifurcation about the juncture of the upper middle third of the esophagus fairly characteristic of malignancy and with only slight dilatation proximally." (Drs. Sherwood Moore and Wendell Scott).

Esophagoscopy for dilatation was done eight days later, and biopsy made then showed general deterioration of malignant tissue. He was generally so improved with easier swallowing that he took adequate liquid diet by mouth, refusing to use the gastrostomy tube.

He reentered hospital December 15, 1940, general external radiation was begun, over back and sides, continued daily for one week. Esophagoscopy was done on December 24, dilating lumen with french catheters, up to size No. 22. This was repeated ten days later, passing the dilators with ease beyond the constriction. Nourishment was easier after each dilatation and never did he show unfavorable reactions following.

Beginning about January 5, 1941, he had constant pain in left shoulder and neck. Such pains began also in the epigastrium and back at margin of the left ribs. February 9. At this phase his lack of interest in food increased, the esophagus seemed totally obstructed. His weakness and loss of weight increased, in spite of adequate feedings by the tube. Evidence of intestinal obstruction appeared on March 21, and he expired March 22, 1941. Autopsy showed extensive periesophageal carcinoma, with metastases to lymph glands of abdomen and to the liver structure.

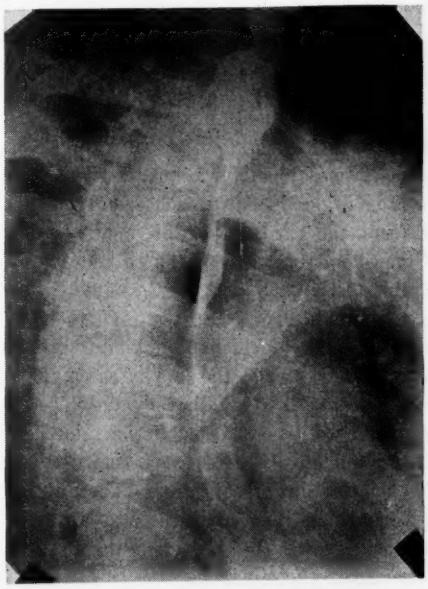


Figure 5. Additional Case: 9-26-33: F.R., "The fluoroscopic examination of the oesophagus by means of a thick barium paste now shows a distinct narrowing beginning above the sterno-clavicular area, and extending several inches below this point. The character of the constriction is rather smooth and is not distinctly typical of carcinoma. The aortic vessels about the arch show distinct saccular dilatation accompanied by expansile pulsation indicating aneurysm. In view of the aneurysm findings of possible leutic origin, the constriction of the oesophagus must be considered also of possible luetic pathology. (Dr. E. C. Ernst).
Autopsy: Aneurysm of the Aorta. Luetic Esophagitis. Carcinoma of Oesophagus, Middle Third.

It is of interest to note the length of time that elapses before patients have a definite examination. The patient himself exercises a certain option, and as noted by Cooper[7] the medical clinic adds to this delay. In the case of cancer

of the stomach he found the patients delay was six months to which four months was added while actually under observation.

In this series of 106 cases, only four were examined during the first month of symptoms, and 57 seen during the first five months. The remainder were scattered through six months to two years of complaint history.

The occurrence in decades of life corresponds to previous reports, i.e., 4 cases in fourth decade, 8 in the fifth, 32 in the sixth and 42 in the seventh, the usual age peak. There were 17 in the eighth decade and two in the ninth. The preponderance of males was 92 to 14 or 5.5 to 1 the usual stated ratio being about 5 to 1 (Bird).[8]

COMMENT:

In addition to the 106 cases of carcinoma primary in the esophagus there were several cases in which the esophagus was invaded by cancer of adjoining organs — two each from the larynx, and from the cardiac portion of the stomach, and one from the trachea. These had symptoms referable to the primary growth, namely, hoarseness and cough from the larynx and trachea, and tarry stools and epigastric pain from the stomach. One year later one of the larynx cases after apparent cure with x-ray therapy developed dysphagia due to a metastasis in the middle third of the esophagus with pearl formation typical of the original laryngeal lesion. There were two cases in which the esophagus was invaded by metastatic growth — one from the breast and another from the stomach. One case with x-ray diagnosis of carcinoma of esophagus but with no esophagoscopy or biopsy, received one dilatation only, yet was well two years later; this is obviously something other than cancer of the esophagus and shows the fallibility of x-ray alone.

The average survival of the 48 cases with date of death reported was 7.6 months; hence, the short duration of this disease in a majority of cases is shown and the urgent need for prompt action by both patient and doctor.

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THE TREATMENT OF PNEUMONIA

WILLIAM L. WINTERS, B. S., M. D.,
WAYNE W. FOX, M. D.
AND
RENO ROSI, M. D.
With the Technical Assistance of
DOLORES LAMMERS, B. S.
CHICAGO
From the ROBERT BRUCE PREBLE RESEARCH LABORATORY,
Department of Medicine Northwesern University Medical School and the
Department of Medicine, Cook County Hospital

The purpose of this paper is to report our experience with sulfapyridine, sulfathiazole and the combined chemo and serotherapy in the treatment of 460 cases of lobar pneumonia in adults seen during the period from January 15, 1939 to June 1, 1940. Many reports have appeared in the literature during the past two years on the use of these methods of treatment. All have substantiated the observation that the mortality rate has been markedly reduced. (25-30% to below 15%)

In this study the clinical diagnosis of pneumonia was established by the history and physical examination. An x-ray examination was obtained on approximately 60% of the patients. This confirmed the clinical diagnosis in all patients so studied. The blood of all patients was cultured in buffered dextrose heart infusion broth. Routine sputum examination and typing by the Neufeld capsule swelling reaction was made immediately after admission; and all sputums which failed to give a direct Neufeld reaction were inoculated into the peritoneal cavity of a mouse. If sputum could not be obtained and blood culture yielded no growth in 18-24 hours, lung puncture was performed. All laboratory work was repeated on those patients who failed to respond within 48 hours. Blood counts were made on admission and on alternate days until

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the patient recovered. A microscopic test for the homologous agglutinin in the blood and Francis (SSS) Test were made frequently in about 40% of the patients.

The dose of sulfapyridine used throughout this study was as follows: an initial dose of 2 grams was given and repeated in two to four hours. A maintenance dose of one gram every four hours day and night was then given, and continued until the temperature dropped to normal and remained so for two more days. From this point, one gram was given every six hours. The drug was stopped when the patient developed signs of active immunity and showed signs of clinical recovery. As an index of active immunity considerable reliance was placed on the Sabin agglutination test. We felt that sulfapyridine should be continued until a strongly positive agglutination with the patient's serum against homologous antigen appeared.[1,2] The average daily dose in this series was 5 grams. The average total dose per patient was 31.5 grams. The largest amount of drug given to one individual was 122 grams in 22 days. The dose of sulfathiazole used in this series was similar to that of sulfapyridine. An initial dose of 4 grams was given and then followed by one gram every four hours, day and night. Cessation of treatment was determined by the presence of a strongly positive agglutination test against homologous antigen. The free sulfapyridine or sulfathiazole blood levels were determined within 24 hours after treatment was instituted and then repeated each alternate day.

The solutions of sodium sulfapyridine or sodium sulfathiazole that were used intravenously, were prepared just before administration by dissolving 5 grams in 100 cc. of distilled water or physiological saline solution that had been previously brought to a temperature just below boiling point. The fluid was given intravenously at a rate of 5 to 10 cc. per minute; or if the patient was getting parenteral fluids the solution was injected into the tubing of the infusion set. In some instances when sodium sulfathiazole was used, it was mixed with the fluid in the infusion flask. One dose given parenterally usually sufficed. If additional drug was necessary, it was given in 12 to 24 hours.

Table I summarizes the type incidence, bacteremic incidence and mortality rate of all the pneumonias treated. Of the 443 patients with pneumococcic pneumonia there were 26 specific

TABLE 1

		A. SULFAPYRIDINE				B. SULFATHIAZOLE				C. S. P. — SERUM				D. SERUM				E. TOTAL SERIES			
		T.	D.	B.	D.	T.	D.	B.	D.	T.	D.	B.	D.	T.	D.	B.	D.	T.	D.	B.	D.
TYPE	I	69	7	19	4	7	—	3	—	6	2	2	2	5	—	1	—	87	9	25	6
	II	75	10	24	9	18	3	7	2	15	4	8	3	6	—	3	—	114	17	42	14
	III	32	1	2	1	1	—	—	—	3	1	1	1	—	—	—	—	36	2	3	2
	IV	13	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	13	1	—	—
	V	13	1	2	1	2	—	1	—	—	—	—	—	—	—	—	—	15	1	3	1
	VI	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—
	VII	42	2	6	1	9	—	1	—	4	1	1	1	1	—	—	—	56	3	8	2
	VIII	36	2	4	1	1	—	—	—	1	—	—	—	2	1	—	—	40	3	4	1
	IX	3	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	1	—	—
	X	1	—	—	—	—	—	—	—	1	1	1	1	—	—	—	—	2	1	1	1
	XI	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XII	5	—	2	—	1	—	—	—	—	—	—	—	—	—	—	—	6	—	2	—
	XIV	7	—	3	—	1	—	—	—	—	—	—	—	—	—	—	—	8	—	3	—
	XV	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XVI	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XVII	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—
	XVIII	5	—	2	—	2	1	1	—	—	—	—	—	—	—	—	—	7	1	3	—
	XIX	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—
	XX	2	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	1	—
	XXII	4	2	1	1	—	—	—	—	—	—	—	—	—	—	—	—	4	2	1	1
	XXIII	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—
	XXIV	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	1	—	—
	XXV	3	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XXIX	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XXXII	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	XXXIII	2	1	1	1	—	—	—	—	—	—	—	—	—	—	—	—	2	1	1	1
Pnc. Uncl.		28	2	—	—	2	2	—	—	—	—	—	—	—	—	—	—	30	4	—	—
Strep.		12	4	5	4	—	—	—	—	—	—	—	—	—	—	—	—	12	4	5	4
Friedlanders		4	2	2	1	—	—	—	—	—	—	—	—	—	—	—	—	4	2	2	1
Staph.		1	1	1	1	—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	1
TOTAL		371	38	75	25	45	6	13	2	30	9	13	8	14	1	4	0	460	54	105	35

Died within 24 hours — 21. Corrected Mortality — 7%.

pneumococcic types represented. Type I accounted for 87, or 19.6%; type II for 114, or 25.7%; type VII for 56, or 12.6%; type VIII for 40 or 9.3%; and type III for 36 or 8.1%. In 30 patients the pneumococcus was recovered but could not be typed with 33 specific types of sera used. These are listed as "pneumococci unclassified" in Table I. Of the 460 patients treated, twelve had streptococcic pneumonias, four Friedlanders and one staphylococcic pneumonia.

Of the group of 354 patients (Table 1A) with pneumococcic pneumonia treated with sulfapyridine alone there were 31 deaths, a mortality of 8.8%. Of the 31 deaths, 15 occurred within 24 hours making a corrected mortality rate of 4.7%. Bacteremia was present in 67 patients and of these, 19 died. Thirteen of the 19 died within 24 hours. The mortality rate of the non-bacteremic group (287 patients) was 4.2%. Eighty patients received intravenous sodium sulfapyridine plus oral sulfapyridine; 5 received intravenous sulfapyridine alone. Early in this study only those patients who were critically ill were given intravenous chemotherapy. When its use was found to be safe, however, it was given routinely to (1) patients who were comatose, (2) all patients whose temperature showed no drop within 18-24 hours after initial dose of sulfapyridine, (3) patients who failed to obtain a level of 4 to 5 mg.% within 24 hours unless the temperature had returned to normal, (4) all patients who could not retain, or refused to take oral sulfapyridine by mouth because of nausea and vomiting, (5) patients with persistent bacteremia and (6) all patients who, after an initial favorable response, had a relapse.

Table 1C analyzes the 30 cases with pneumococcic pneumonia treated with both sulfapyridine and serum. Six different types were represented, but types I, II and VII were isolated in 25 of the 30 patients. Of the 30 patients treated, 9 died. Bacteremia was present in 13 patients, and of these, 8 died. Of the 9 deaths, one occurred within 8 hours of admission, another within 30 hours and a third died of anaphylactic shock during serum administration.

Table 1D analyzes a small group of patients seen early in this study who received serum alone. Fourteen patients comprise the group. Types I and II account for eleven of the 14 pneumococcic types isolated. Only one death occurred. Bacteremia was present in 4 patients. No deaths occurred in the bacteremic group.

Table 1E summarizes all the pneumococcic pneumonias treated (all types of treatment). Of the 443 patients treated there were 47 deaths, a mortality of 10.6%. Twenty-four deaths occurred within 24 hours, making a corrected mortality of 5.5%. Of the 443 patients, 97 (21.9%) were bacteremic, and of these, 35 (36%) died.

An analysis of the fatal cases is of particular value in pointing out the factors for failure. Those influencing the course of a pneumonia, irrespective of treatment, are (1) duration of the disease before treatment, (2) age, (3) bacteremia, (4) extent of lung involvement, (5) alcoholism.

Effect of Delay in Treatment (Table II). Analyzing those patients who received sulfapyridine, the efficiency of treatment that was started after the fifth day of the disease was markedly reduced. Of the 195 patients in whom treatment was begun before the fifth day, 11 died and 47 developed complications; while of 159 patients in whom treatment was begun after the fifth day, 20 died and 74 developed complications. This delay in treatment is probably the most important single factor in prognosis and is rarely if ever controlled by the physician. Further education of the public is the only hope of giving patients adequate treatment earlier in their illness.

TABLE II

DELAY IN TREATMENT
PNEUMOCOCCUS PNEUMONIA SULFAPYRIDINE TREATED

Day of Disease	Number		Died		Complications	
1	16		0		2	
2	47		2		5	
		195		11		47
3	67		5		20	
4	65		4		20	
5	80		6		33	
		159		20		74
Over 5	79		14		41	
TOTAL	354		31		121	

Effect of Age (Table III). The next factor influencing mortality in this study was age. In the group of pneumococcus pneumonias treated, there were 214 patients under 40 years of age, with 8 deaths; and 229 patients over 40 years of age with 39 deaths. In the sulfapyridine treated group, there were 177 patients under 40 with 7

deaths, and 177 patients over 40 with 24 deaths. In the combined sulfapyridine and serum group, there were 13 patients under 40, with no deaths; and 17 patients over 40, with nine deaths. In the sulfathiazole treated group, there were 15

TABLE III

EFFECT OF AGE ON OUTCOME OF PNEUMONIA

AGE	S.P.	S.P., SER.	S.T.	SER.	TOTAL	DIED
Pneumococcus pneumonia.						
0-19	22- 0	——	2-0	1-0	25	0
20-29	63- 3	6-0	6-0	3-0	78	3
30-39	92- 4	7-0	7-1	5-0	111	5
40-49	78- 4	7-5	12-1	4-1	101	11
50-59	57-12	4-1	7-2	1-0	69	15
60-69	34- 5	6-3	8-1	——	48	9
70-	8- 3	——	3-1	——	11	4
0-40	177- 7	13-0	15-1	9-0	214	8
40-	177-24	17-9	30-5	5-1	229	39
Others: 12 Streptococcus 4 Friedlander One Staphylococcus						
0-40	12- 4	——	——	——	12	4
40-	5- 3	——	——	——	5	3
TOTAL	371-38	30-9	45-6	14-1	460	54

patients under 40, with one death; 30 patients over 40, with five deaths. Of 17 patients who were treated with sulfapyridine and whose etiology was streptococcus, B. Friedlander, and staphylococcus, 12 were under 40 years of age, with 4 deaths; and five were over 40, with 3 deaths.

Extent of Lung Involvement (Table IV). The extent of lung involvement is also of prognostic value. In the whole series 333 patients had one lobe consolidated, and there were 24 deaths (7.3%); while in 127 with two lobes or more consolidated, there were 30 deaths (23.6%). Usually the extent of involvement parallels the duration of the disease before treatment is begun and is therefore controllable only through obtaining medical care earlier in the course of the illness.

TABLE IV

EXTENT OF LUNG INVOLVEMENT (ALL CASES)

LOBES	TOTAL Pnc.	TOTAL Others	DIED Pnc.	DIED Others	
1 LOBE	323	10	21	3	7.2%
MORES THAN 1 LOBE	120	7	23	7	23.6%
TOTAL		460		54	11.7%

Effect of Bacteremia (Table I). Invasion of the blood stream by the pneumococcus during the course of lobar pneumonia occurred in 96 of the 443 patients. When it did occur it was a serious prognostic sign as indicated by Table I. In the 354 sulfapyridine treated patients, 67 had positive blood cultures, and of these, 19 died, while of the 287 non-bacteremic patients, there were only 13 deaths. Of 45 patients treated with sulfathiazole, there were 13 with positive blood cultures, and two died. Of the 32 non-bacteremic patients treated with sulfathiazole, 4 died. Thirty patients were treated with combined sulfapyridine and serum. Thirteen were bacteremic with 8 deaths; 17 were non-bacteremic with one death. Failure of a patient to respond within 24-36 hours of treatment with sulfapyridine or sulfathiazole or one of these agents combined with serum was commonly due to an invasion of the blood stream by the pneumococcus.

Effect of Alcohol (Table V). In the Cook County Hospital, as in other large municipal and county hospitals, alcoholism is commonly associated with lobar pneumonia. In this series of 443 patients, 66 gave a history of being moderate to heavy drinkers, and 22 died. Of the 66 alcoholics, 29 had positive blood cultures, and 17 died. Of the 377 non-alcoholics, 32 died. Seventy-six patients in the non-alcoholic group had positive blood cultures and 18 died. Of the 318 non-alcoholics with negative blood cultures, there were 14 deaths.

TABLE V

EFFECT OF ALCOHOL

ALCOHOLISM	TOTAL	DIED
BACT.	29	17 (58.5%)
NON-BACT.	37	5 (13.5%)
TOTAL	66	22 (33%)
NON-ALCOHOLIC		
BACT.	76	18 (23%)
NON-BACT.	318	14 (4.4%)
TOTAL	394	32 (8.1%)

COMPLICATIONS — PNEUMOCOCCIC — PNEUMONIA

443 CASES	BACTEREMIA	NON-BACT.	TOTAL
BACTEREMIA	96	347	—
EMPYEMA	11	3	14
DELAYED RESOLUTION	10	8	18
PLEURAL EFFUSION	4	6	9
JAUNDICE (hepatitis)	4	2	6
LUNG ABSCESS	1	1	2
HYPO-CHROMIC ANEMIA	1	2	3
PAROTITIS	—	1	1
ARTHRITIS	—	1	1

REACTIONS

	SULFA-PYRIDINE 371 CASES	SULFA-THIAZOLE 45 CASES	SULFA-PYRIDINE AND SERUM 30 CASES
NAUSEA & VOMITING	90	4	6
RASH	7	–	–
FEVER	6	–	–
ANEMIA	1	–	1
LEUCOPENIA	4	–	–
NPN RETENTION	4	–	1
HEMOLYTIC ANEMIA	1	–	1
HEMATURIA	1	–	1
CEREBRAL SYMPTOMS	1	–	–

Effect of Treatment on Physical Findings. After 12-36 hours of sulfapyridine therapy a precipitous drop in the temperature usually occurred. This happened in the presence of bacteremia and often in spite of low leucocyte counts and the absence of the homologous agglutinin in the blood. Associated with the drop in temperature there was a slowing of the pulse and the respiratory rate, and the toxicity abated. Although the improvement in the general condition of the patient was quite marked, very little change in the physical signs over the involved lung could be detected. It remained consolidated and seemed to undergo the changes seen in an untreated pneumonia. The agglutinins did not appear in the circulating blood until an average of the tenth day after onset of pneumonia. By then the patient manifested signs of overcoming the infection. Sulfapyridine therapy apparently did not affect agglutinin production. A strong reaction occurred in over 90% of patients tested. In certain patients treated within the first 24 hours of the disease, frank signs of consolidation never did appear and the signs of infection were gone within a week. With favorable response to sulfapyridine therapy, the need for oxygen and other supportive measures was decidedly lessened.

The action of sulfathiazole on the clinical findings were similar to that of sulfapyridine with the one exception, that the early response, as evidenced by a drop in the temperature, pulse, and respiration, was more gradual and often did not occur until 36-72 hours had elapsed. With sulfapyridine, on the other hand, the response took place in 24-36 hours.

Complications. Bacteremia occurred in 96 patients with pneumococcus pneumonia. In several patients it was persistent in spite of active sulfapyridine or sulfathiazole therapy. One patient had five positive blood cultures during 15 days of sulfapyridine therapy, in which time the level of free sulfapyridine in the blood varied from 4.0 mg.% to 7.8 mg.% with an average of 5.6 mg.%. This patient died on the fifteenth day after treatment was begun. Another patient had four positive blood cultures in succession over a period of 11 days, during which time the level of free sulfapyridine in the blood ranged from 1.25 mg.% to 5.25 mg.% with an average of 2.8 mg.%. A negative blood culture was obtained on the thirteenth day of treatment. This patient developed an empyema but eventually recovered.

Empyema was observed in 14 patients and the type pneumococcus isolated from the pus corresponded to that found in the sputum. Eleven of the 14 had positive blood culture. The pneumococcic types represented were Types I, II, III, and VIII. With continued sulfapyridine treatment and repeated chest aspirations the septic course was lessened but all 14 eventually required surgical drainage. Two failed to recover.

Delayed resolution was seen in 18 patients, 10 with bacteremia and 8 non-bacteremic. All were confirmed by x-ray. Clinically, two of these patients appeared entirely well but the others developed fever of from 101° to 103° F. which persisted for several days, then subsided spontaneously by lysis. The rise in temperature often followed an afebrile period of from 2-4 days.

Sterile pleural effusion occurred in 10 patients, four with bacteremia and six with negative blood cultures. The response to continued sulfapyridine and repeated chest aspirations was favorable in all patients. Repeated studies on the pleural fluid failed to yield any organism.

Other complications which occurred were: jaundice six times, pronounced hypochromic anemia three times, lung abscess twice, acute parotitis once, and acute arthritis once.

Sulfapyridine Reaction. Anorexia, nausea and vomiting were the most common toxic reactions encountered during sulfapyridine treatment but were seldom severe enough to warrant cessation of the drug. Nausea and vomiting were present in 96 patients. In ten, the vomiting was of such a degree that the oral drug was stopped and intraveous sodium sulfapyridine was substituted. In about one third of the patients, sodium bicarbonate was given and it seemed to have little effect on the prevention of the above gastrointestinal symptoms. The administration of oxygen has been used successfully in the control of nausea and vomiting due to sulfapyridine[3] but in our study these symptoms persisted in spite of oxygen administration.

The incidence and severity of nausea and vomiting with sulfathiazole was decidedly less than that of sulfapyridine and occurred in only

four of the 45 patients treated. Cessation of sulfathiazole treatment was not warranted by the degree of nausea and vomiting present.

One of the most distressing sulfapyridine reactions was fever and rash. In six patients a sulfapyridine fever was diagnosed. It usually occurred after 5-7 days of treatment, often after an afebrile period when the patient appeared clinically well. The duration of the fever varied from 2-4 days and ranged from 101° to 103° F. In two patients the fever developed when sulfapyridine was resumed after an interruption of 2-3 days which followed the initial course of several days. In all patients who showed fever and one that did not, a maculopapular morbilliform rash appeared over the torso, arms, legs, and occasionally on the mucous membranes of the palate and pharynx. The presence of fever without clinical evidence of spread of the infection, complications, and a normal leucocyte count points to a drug fever. Both the fever and the rash disappeared after the drug was stopped and fluids forced.

Anemia of the hypochromic variety occurred six times; leucopenia occurred four times; nitrogen retention occurred four times; hemolytic anemia and hematuria occurred twice, and cerebral symptoms once. All responded as soon as the drug was stopped and fluids given freely.

SUMMARY AND DISCUSSION

During the 17 month period, January 1939 to June 1940, 460 patients with pneumonia were treated. Mortality was 11.7%. Of these 460 patients, 443 had pneumococcic pneumonia. Forty-seven (10.6%) died. Deducting the number of deaths that occurred within 24 hours of admission (21) gives a corrected mortality of 7%. Four types of treatment were used. Sulfapyridine alone forms the largest group. Sulfathiazole, sulfapyridine and serum combined, and serum alone, comprise small groups.

Analysis of this group shows that sulfapyridine was effective against all types of pneumococcus encountered (26 types) and that its use is safe. No deaths could be attributed to the drug. Mild toxic reactions occurred frequently. The number of patients treated with sulfathiazole is too small to be of statistical value. The drug is effective against the pneumococcus, and the incidence of reactions, particularly nausea and vomiting, is definitely less than with sulfapyridine. The initial response to this drug is delayed longer than with sulfapyridine. Our experience indicates that sodium sulfapyridine and sodium sulfathiazole can be used safely intravenously, and are of distinct value in establishing a therapeutic blood level immediately. They should be kept in mind by the physician treating pneumonia as an efficient therapeutic agent.

The effectiveness of serum has been definitely established in the past and further study on this method of treatment was not stressed in this series. The patients who received serum, however, showed a very satisfactory clinical response.

A study of the fatal cases indicates that the most important factor in prognosis was delay in treatment. Next in order of importance were age, extent of lung involvement, bacteremia, and alcoholism. During most of this study serum was not administered until it appeared obvious that sulfapyridine alone was inadequate as indicated by continued fever and prostration, persistent bacteremia, spread of the pneumonia to other lobes, or the inability to develop immune bodies. Toward the end of this study it was felt that under certain conditions intravenous sulfonamide therapy or serum plus sulfonamide therapy should be considered. These conditions were: duration of the disease four or more days; age over 40; involvement of more than one lobe; alcoholism and evidence of beginning septic complications or presence of associated diseases. We also feel that patients whose admission blood cultures are positive should be considered candidates for intravenous sulfonamide therapy, or serum, unless there has been a striking improvement in their condition in the interval elapsing from the time the blood culture is drawn to the time the positive blood culture is observed.

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EARLY DIAGNOSIS OF BRAIN TUMORS

A. Earl Walker, M.D.
CHICAGO

The time honored clinical triad of brain tumor — headache, papilledema, and vomiting — has long since been shown to be indicative only of intracranial hypertension, and hence usually a late manifestation of intracranial tumor.

Because of better methods of examinations and the fact that physicians now realize the frequency of brain tumors, and hence consider the possibility more often, in recent years more and more tumors have been recognized before they caused intracranial hypertension. In 1934 Ley and Walker[7] analysed a group of 230 intracranial tumors from the service of Dr. Percival Bailey at the University of Chicago Clinics. In this series, 69.0 per cent. had papilledema. In the last one hundred cases of verified brain tumor (May 1937 to November 1939) seen at the same clinic, only 54 per cent. had choked discs. The difference in the incidence of this finding between these two series is quite significant, for the distribution and localization of the tumors in the two series was much the same. The reality of this difference is confirmed by the fact that in the first group headache (usually an indication of intracranial hypertension) occurred in 74.9 per cent. of the cases and in the second series in only 66 per cent.; furthermore, the incidence of vomiting was markedly reduced in the later series (48 per cent. in the first group; 39 per cent. in the second). This, therefore, indicates that brain tumors are being suspected and diagnosed before they produce intracranial pressure and hence in most instances on different bases than formerly. Before discussing some of the more recent developments in diagnostic technique, let us briefly review some of the early manifestations of brain tumor.

As Bailey[1] stated, there is only one cardinal symptom of tumor of the nervous system — a non-febrile, steadily increasing alteration in nervous function. But not all tumors progress in this manner — some are ushered in by sudden and startling nervous phenomena. However, practically all intracranial neoplasms manifest themselves by an alteration of nervous function. The peculiar type of nervous function which becomes impaired, will obviously depend upon the locus of the tumor, for pathological processes in different parts of the brain produce distinct clinical syndromes.

It is obviously impossible to present all the initial symptoms of brain tumor, for such a discussion would entail a review of the entire physiology of the brain, since the first manifestation of a tumor may be either an excitation or depression of the function of any part of the brain. Some of the more common manifestations merit attention.

Headache. Although frequently evidence of intracranial hypertension, headache has occurred in 21 per cent. of the last one hundred verified brain tumors without papilledema or intracranial hypertension as determined by spinal fluid manometric readings. In general it is by far the most common initial complaint, although in children under fifteen years of age, vomiting has a slightly greater frequency. The headache is not characteristic, although frequently it is said to be more severe in the morning. It is aggravated by coughing, sneezing, or stooping. The localization of the headache is usually of no assistance in determining the position of the tumor, although occasionally the pain is definitely referred to the site of the tumor. The headache of brain tumor is often no different than that of many other conditions. The symptom *per se,* then, does no more than invite the physician to examine carefully the nervous system for more definite evidence of intracranial pathology. It is unusual for headache alone to be the only manifestation of a brain tumor.

Vomiting. Although in most instances like headache, vomiting is evidence of intracranial hypertension, it may occasionally be a symptom of definitely localizing value. As Bailey, Buchanan and Bucy[2] emphasize, persistent vomiting in children, unassociated with headache, is usually an indication of irritation of the floor of the fourth ventricle, most frequently resulting from a glioma of the pons or a tumor of the fourth ventricle.

This symptom is more common in children, and characteristically occurs early in the morning, even before breakfast. Although usually associated with nausea the patient is commonly quite prepared after the attack to eat another breakfast. This is not invariably true, however, and in some instances a severe anorexia, result-

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From the Division of Neurological Surgery, the University of Chicago, Chicago, Illinois.

ing in emaciation, is associated with the nausea and vomiting. The vomiting is rarely projectile but may be explosive. It seems to relieve the headache that frequently accompanies it. In children, vomiting may occur day after day, but in adults it is usually less frequent unless the patient is on the verge of coma from intracranial hypertension. Repeated attacks of vomiting without adequate cause should always lead to the suspicion of intracranial neoplasm.

Convulsions. This is a very frequent initial symptom of a brain tumor. In the first series of 230 cases of brain tumor, 17.9 per cent. of the patients presented it as their first evidence of intracranial pathology. It is practically always evidence of a supratentorial tumor, although patients with cerebellar tumors are known to have generalized seizures. More common, however, are the so-called cerebellar fits or tonic opisthotonic attacks, in which the head is retracted, the back arched, and the limbs tonically extended. Respiration may cease in such attacks and death ensue. In children a fit does not cause undue alarm to parents or physician since idiopathic epilepsy is so common at that age, but in adults the occurrence of a generalized convulsion is a matter of grave concern. After twenty years of age the incidence of idiopathic epilepsy is less than 20 per cent. and at forty years only five to six per cent. Every adult having a seizure for the first time should be suspected of harboring an organic brain lesion, possibly a tumor, until proved otherwise. Statistically such an individual has about equal chances of suffering from cerebral vascular disease, cerebral lues, brain tumor, or no demonstrable organic brain pathology.[9] And the examination should not only be the routine clinical neurological checkup, but should also include electroencephalography and possibly air encephalography, for many of these cases will have no demonstrable clinical evidence of cerebral dysfunction at the time of their first attack, and yet harbor pathology which may be shown by one of the above mentioned special methods.

The type of convulsive attack and particularly its beginning is of especial value for a localizing diagnosis. An epileptic attack beginning with a motor or sensory aura in one part of the body generally indicates a focal lesion in the cerebral cortical representation of that portion of the body. For example, a forty-year old housewife, who suffered from attacks beginning with a spasm of the jaws and clonic movements of the right side of the face, was found to have a small meningioma overlying the face representation in the left motor area. Similarly an attack beginning with twitching of the foot, then of the leg, spreading to the thigh and ipsilateral arm (Jacksonian epilepsy) is typical, although not pathognomonic of a parasagittal meningioma. Somatosensory, auditory, visual, olfactory and gustatory aura have similar localizing significance to their respective cortical representations. The tonic opisthotonoid seizure associated with tumors of the posterior fossa has already been mentioned.

Failing vision. Impairment of vision as an initial symptom of brain tumor is not infrequent (13.7 per cent.). It is almost always associated with a lesion above the tentorium and usually with a tumor pressing upon the optic nerves, chiasm or tracts. A careful examination of the visual fields is often of great value in the accurate localization of the lesion.

Progressive paralysis. In spite of the fact that a steady progressive loss of cerebral function is the one cardinal symptom of intracranial neoplasm, a progressive paralysis is not nearly as common an initial symptom as might be imagined. A neoplasm involving the motor areas usually gives rise to a convulsive seizure before the weakness becomes prominent. Although a progressive paralysis usually indicates a lesion of the cerebral hemispheres, a pontine or mesencephalic tumor occasionally gives rise to such a condition.

This symptom — a slowly progressive paralysis — is not pathognomonic of a tumor. It may be produced by many other conditions — spinal cord neoplasm, amyotrophic lateral sclerosis, Pick's disease, or other degenerative conditions. In these conditions epileptic attacks with or without a focal beginning are rare, so that if a progressive weakness is associated with convulsive seizures a cerebral neoplasm is likely; if unassociated with such attacks, a degenerative disease is more apt to be the etiological factor.

Staggering gait. Less frequently the patient complains of an unsteady gait and almost invariably the tumor is found in the posterior fossa. Such tumors being more common in children, this difficulty is not frequently seen in adults suffering from cerebral neoplasms, al-

though acoustic neurinomas do cause such a disturbance.

Other initial symptoms of brain tumor. Only a few of the many complaints that may be the first warning of a brain tumor have been mentioned. Many others might be cited: the tinnitus and deafness, which exists for many years before the patient seeks relief from an acoustic neurinoma; the progressive exophthalmus, which characterizes the sphenoidal ridge meningioma; the progressive enlargement of the hands and feet with loss of pep and libido, which indicate pituitary dysfunction; and the somnolence and obesity, which accompany tumors in the region of the third ventricle. Not uncommonly a sudden hemiplegia resulting from thrombosis or hemorrhage in or about a tumor will herald its presence. This is particularly true of the onset of the malignant cerebral gliomas of middle life.

Clinical signs of brain tumor. A careful neurological examination of the brain tumor suspect will usually reveal some evidence, even if only slight, of dysfunction of the nervous system. The nature of this defect will depend upon the site of the tumor and hence may involve any of the processes that the brain normally performs.

Spinal fluid findings. The examination of the spinal fluid in most cases of brain tumor is of little value in assisting in a diagnosis, and where intracranial hypertension exists is definitely hazardous. Under the latter circumstances a lumbar puncture may precipitate a hemorrhage into the tumor or cause medullary compression by allowing a herniated cerebellar pressure cone to jam further into the foramen magnum. Because of these dangers and the usually meager information gained, lumbar puncture is not advocated when papilledema is present or when a cerebellar tumor is suspected. When choked discs are not present, lumbar puncture in patients with brain tumor may reveal increased pressure, usually of a moderate degree. (I have seen a pressure of 400 mm. of spinal fluid, twice the normal, in the absence of papilledema.) But frequently the spinal fluid is under normal or even a subnormal pressure (in one patient with a large left temporal glioblastoma, 50 mm. of spinal fluid, and in another comatose patient with a left temporal lobe mixed astrocytoma and oligodendroglioma 95 mm. of spinal fluid). Hence a normal or subnormal spinal fluid pressure, even in a comatose patient does not eliminate the possibility of an intracranial tumor. The protein as determined by Pandy's test and quantitatively is frequently increased, particularly if the tumor is near the ventricular wall or is an acoustic neurinoma. Cells up to several hundred may be present in the spinal fluid, but are practically always accompanied by an increased protein. Occasionally the colloidal gold curve is abnormal — usually paretic in type — especially in the case of an acoustic neurinoma. The chlorides and sugar are usually normal. It is, therefore, obvious that the value of a lumbar puncture in cases of brain tumor is not so much in confirming the diagnosis as in eliminating other possible etiological factors such as chronic meningitis, cerebral lues, etc.

Electroencephalographic manifestations. As early as 1875 Caton[5] demonstrated the presence of changing electrical potentials in the brain. It was Berger[3], however, over fifty years later, who showed their importance and popularized the study of brain potentials or, as they are commonly termed, "brain waves." When pathological conditions, particularly tumors, are present in the brain, the electrical potentials are frequently interfered with, thus giving us another means by which we may determine the presence of an intracranial neoplasm.

The electroencephalogram is made by leading off the electrical potentials from different points by small silver discs applied to the intact scalp, amplifying them and recording them on an ink writer. Usually the potentials from several areas are recorded simultaneously. The procedure has the distinct merit that it does not inconvenience the patient in any way, and causes no after effects. Normally in man, waves having a frequency of 7.5 — 11 per second (alpha waves), 14 per second and 18 - 35 per second are found. Certain other frequencies occur in physiological states such as sleep, or in pathological conditions. Case and Bucy[4] have pointed out that intracranial lesions are indicated by the presence of one or another of the following electroencephalographic phenomena in one or more of the cortical areas:

1. Very slow swings 1 - 2 in five seconds.
2. Slow waves ½ to 3 per second.
3. Spikes and saw tooth phenomena.
4. Localized electrical manifestations of epileptic seizures.
5. Absence of or marked difference in alpha

waves on the two sides.

6. Marked diminution or localized absence of electrical activity.

The slower waves are more commonly seen in degenerative or atrophic conditions, whereas the 1 - 3 per sec. waves, spikes, etc. are apt to be associated with neoplastic diseases of the brain.

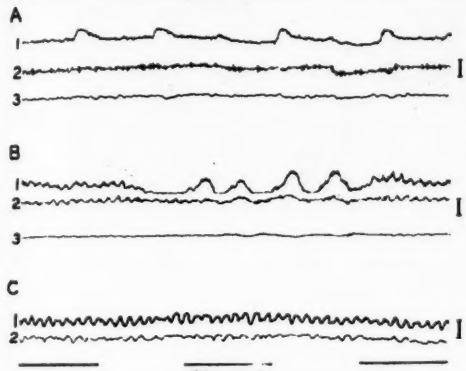

Figure 1.

Figure 1. The typical electroencephalogram of a normal person. (Leads — 1. right frontal, 2. right temporal, 3. right occipital.) In the first lead the humps are due to blinking movements and should not be confused with delta waves. In the second lead, the small spikes are the result of muscular discharge and must be differentiated from epileptic manifestations. In the third lead the indistinct alpha waves are probably to be associated with the fact that the patient had his eyes open.

B. An electroencephalogram of a patient suffering from a left frontal lobe tumor. (Leads — 1. left frontal, 2. right frontal, 3. right temporal.) Large 2 per second delta waves are pronounced in the left frontal lead, and absent in the other leads. The alpha rhythm is evident in both frontal leads.

C. The electroencephalogram of a patient with a left temporo-occipital lobe tumor showing the disparity of the alpha waves on the two sides. (Leads — 1. right occipital, 2. left occipital.)

The time in seconds is indicated at the bottom of the tracings; the vertical lines at the right indicate the deviation produced by 100 millivolts. (These electroencephalograms are reproduced by the courtesy of Dr. Theodore J. Case.)

Let us examine the recent series of brain tumors to determine the frequency with which electroencephalographic disturbances are encountered. In exactly one half or fifty of the cases, electroencephalographic records are available. In twenty-four cases the findings were sufficiently clear to warrant an interpretation as to location of the tumor. In eighteen of these cases a correct electroencephalographic diagnosis was made, and in five other cases it was suggested. In only one case — an early one — was an inaccurate localization made, and that error might have been avoided at the present time. In twenty-six cases, ten of which were tumor of the posterior fossa, there was insufficient evidence for a diagnosis. It should be pointed out that, with the present techniques, cerebellar lesions have rarely shown alterations in the electroencephalograms.

Although the localization of the majority of these tumors was apparent from the clinical examination, in twenty-four of the fifty cases air studies were made because the examiner was not certain of the exact location of the tumor. In eleven of these cases the electroencephalogram correctly localized the lesion. In one of these cases, even after ventriculography, the localization of the tumor was erroneous and in spite of the electroencephalographic findings the posterior fossa was explored when a frontal lobe tumor was present.

The electroencephalogram is, therefore, a valuable adjunct in the diagnosis of intracranial lesions. It is not diagnostic in every case, but when positive evidence of neoplasm is present the findings are quite reliable. Since the procedure causes the patient no inconvenience, it may be repeated on several occasions, increasing the changes of a correct localization.

Roentgenological Manifestations of Brain Tumor. Roentgen ray examination of the skull is another important diagnostic instrument. Many evidences of intracranial neoplasm are so revealed. I need only mention the enlargement of the pituitary fossa in acromegaly and chromophobe adenomas of the hypophysis, the suprasellar calcifications in craniopharyngiomas, the intracerebral calcifications in oligodendrogliomas and astrocytomas, the erosions and increased vascularity of the calvarium in certain meningiomas, the enlargement of the internal acoustic meatus and optic foramen with acoustic neurinomas and gliomas of the optic nerve respectively, and the bony proliferations overlying meningiomas, especially of the sphenoidal ridge. These changes are well known, usually easily recognized and practically pathognomonic of the respective type of brain tumor. Roentgenograms of the head may assist in the diagnosis of brain tumor by revealing displacements of the calcified pineal gland, for it normally lies exactly in the mid-line of the skull.

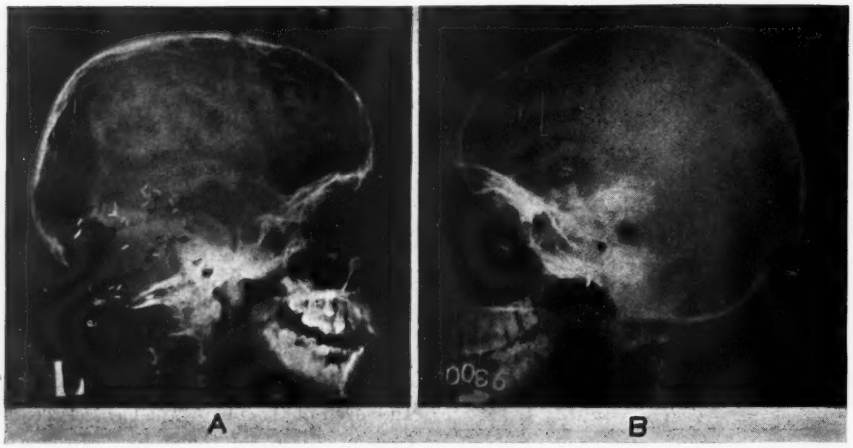

Figure 2.

Figure 2. Roentgenograms of the skull to show:

A. Convolutional atrophy of the calvarium and sutural diastasis, the result of a large cerebellar astrocytoma, the size of which can be estimated by the silver clips used to control bleeding at the time of operation.

B. A large calcified suprasellar tumor.

One should not forget that an x-ray photograph of the chest may clear up a puzzling cerebral syndrome by revealing primary or secondary pulmonary tumors when only cerebral manifestations are present.

Encephalography and ventriculography. Valuable as the roentgenological examination of the skull is, it becomes still more important when it is made after the cerebrospinal and/or ventricular fluids have been replaced by a gas allowing visualization of the shape, size and position of the brain substance.

When Dandy[6] introduced the method of ventriculography in 1918, he gave to the neurologist and neurosurgeon one of the most useful diagnostic methods now at his disposal. Since that time the normal appearance of the cerebrospinal fluid pathways and ventricular system have been described in detail. Ventriculography is the term applied when the cerebral ventricles are directly punctured and the fluid replaced by a gas; encephalography denominates the replacement of the spinal and ventricular fluid through a lumbar puncture needle. This latter procedure should only be carried out when there is no intracranial hypertension; if performed in the presence of increased pressure, there is danger of medullary compression due to further herniation of a cerebellar pressure cone. Furthermore, encephalography under such circumstances is often useless as the ventricles frequently fail to fill with the gas.

Roentgenograms made after these procedures will demonstrate the size, position and shape of the cerebral ventricles and, in the case of encephalography, of the basal cisterns and the subarachnoid space. Tumors give rise to displacement, distortion or incomplete filling of the ventricular system and usually are readily diagnosed by this means. Only when the filling of the ventricles is incomplete does the interpretation of the ventriculograms become difficult. The ventricular abnormalities shown by this method are probably the most reliable single indication of brain tumor. It is true that occasionally a brain tumor may exist in the presence of an apparently normal ventricular system, but this is uncommon when ventricular filling is complete.

Air studies of the ventricular system are not necessary in every case. The clinical diagnosis corroborated by electroencephalographic phenomena or abnormalities in the regular roentgenograms of the skull were sufficient in fifty-five of the last hundred cases of brain tumor, ventriculography or encephalography being performed in 45 per cent. of the cases. In the earlier series, air studies were only made in 20 per cent. of the cases. The marked increase in the number of air studies is largely due to the fact that the cases are being seen much earlier than a

few years ago — so early, in fact, that it is impossible to be certain by clinical methods whether one is dealing with an inflammatory, degenerative, or neoplastic condition of the brain. As mentioned previously, the lowered incidence of papilledema is another indication that these cases are being seen earlier, before intracranial hypertension develops and before localizing signs are definite.

Arteriography. Although Moniz[8] suggested the injection of sodium iodide into the carotid artery for the visualization of the cerebral circulation more than ten years ago, the severe complications resulting prevented its general use. Subsequently he introduced the use of thorotrast for injection for this purpose and it has been found to be unattended by serious complications. As a result, cerebral arteriography is slowly gaining popularity although the possible late effect of the thorium on the liver, spleen, and kidneys has impeded the universal acceptance of this procedure.

It does enable a visualization of the tumor by demonstrating the abnormal vessels within the tumor, and the displacement of the normal cerebral vessels. There is some evidence to suggest that the arteriographic pattern of the different types of brain tumors are more or less characteristic, and the hope has been expressed that it might be possible to diagnose malignant tumors of the cerebral hemisphere by this method and thus eliminate some of the frequently futile craniotomies. Whether these hopes will be realized remains for future research to decide. The method is particularly useful with blood-vessel tumors and aneurysms.

DISCUSSION

In this brief review of the clinical, electroencephalographic and roentgenological manifestations of brain tumors, the manifold ways in which neoplasms arising within the intracranial cavity present themselves have been indicated. Early in their course many brain tumors initiate excitatory phenomena, such as convulsions, and produce little evidence of other dysfunction of nervous tissue. It is at this time that their diagnosis is so difficult. Later, when the impairment of nervous function is more prominent, and the neoplasm has increased the bulk of the intracranial contents sufficiently to cause hypertension and choked discs, the diagnosis becomes easier. This is evidenced by the fact that of those cases having choked discs only 38 per cent. required air studies whereas in 54 per cent. of the cases without papilledema, ventriculography or encephalography was necessary. In this latter group of cases in which the evidence of nervous dysfunction is slight, it is essential to eliminate other possible etiological factors, for in most instances vascular lesions, inflammations and degenerations could conceivably give rise to the same clinical picture. Yet it is not desirous to submit all patients suffering from vascular insults, inflammations of the nervous system and degenerations to air encephalography, for the

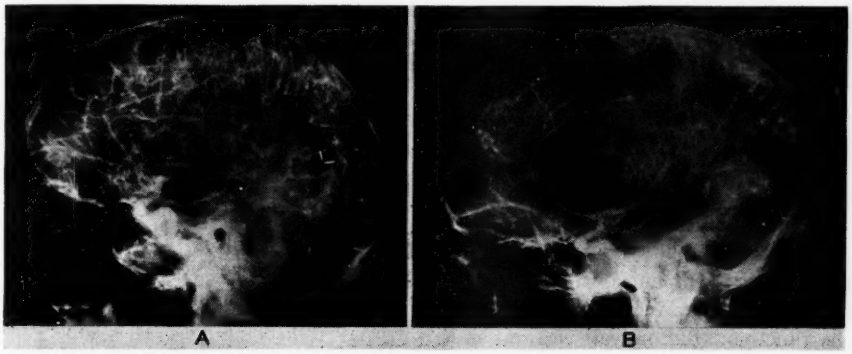

Figure 3.

Figure 3. Cerebral arteriograms:
A. Normal.
B. Patient with a large left frontal lobe cyst (probably glioblastomatous). Note the posterior displacement of the sylvian vessels and the avascular area just anterior to these vessels in the inferior frontal region. The tangled vessels in the anterior part of the frontal lobe probably are within the tumor.

latter requires hospitalization, gives an annoying headache for several days and it not without danger. And in most instances this is unnecessary since by a carefully elicited history, detailed neurological examination and the examination of regular roentgenograms of the skull the majority of such cases can be correctly diagnosed. If a doubt as to the proper diagnosis still exists, electroencephalography — a harmless and simple procedure — frequently will suggest the etiological factor. If uncertainty persists, repeated complete examinations at month or six weeks intervals may dispel the doubt, for the progress and course of most diseases of the nervous system is characteristic. For the rare case, which has not disclosed its identity by this time, lumbar puncture and air encephalography may be necessary. By this means, with as little discomfort to the patient as possible, it becomes possible to make an early correct diagnosis of a slight nervous dysfunction which may be the initial evidence of a brain tumor.

SUMMARY

The only cardinal symptom of a brain tumor is a non-febrile, steadily increasing alternation of nervous function. But not all cerebral neoplasms progress in this manner — some are ushered in by sudden and startling nervous phenomena. Commonly headache, vomiting, convulsions, failing vision, progressive paralysis, staggering gait, are the initial manifestations of a brain tumor. A careful neurological examination will usually reveal some evidence of dysfunction of the nervous system. When the tumor becomes large enough to cause intracranial hypertension either from blockage of the spinal fluid pathways or from its mass, papilledema usually develops and localizing signs are more common. The spinal fluid rarely gives valuable evidence of tumor but examination of it is useful in eliminating other possible etiological factors. Electroencephalographic studies are extremely valuable and may be made without inconvenience to the patient. Roentgenograms of the skull frequently show evidence of a tumor. Encephalography and ventriculography are valuable adjuncts but are only necessary when clinical and simpler technical diagnostic aids have been exhausted.

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SPONTANEOUS HEMORRHAGE INTO THE SHEATH OF THE RECTUS ABDOMINIS MUSCLE

E. P. Coleman, M.D., F.A.C.S. and D. A. Bennett, M.D.

CANTON

Spontaneous hemorrhage into the rectus abdominis muscle is difficult to diagnose and a careful review of medical writings reveals that in very few instances has the correct diagnosis been made before operation. As a rule, it is impossible to distinguish it from other abdominal conditions. A review of the literature indicates that the true condition is usually overlooked, and most of these patients have been subjected to laparotomy with the mistaken diagnosis of some acute intra-abdominal condition. These diagnoses have included appendicitis, a tumor, ectopic gestation, an incarcerated hernia, mesenteric thrombosis, intussusception, volvulus, ventral hernia, gall-bladder disease, or fibroid tumor of the uterus. It is obvious, therefore, that recognition of the symptoms indicating spontaneous hemorrhage into the rectus muscle is extremely important when considering the differential diagnosis of abdominal pathology.

Furthermore, while spontaneous hemorrhage into the rectus muscle is far from common, it should not be regarded as of exceedingly rare occurrence. In 1882, Maydel reviewed all medical literature from 1809 to 1880 and collected 14 cases that occurred before 1880. It is his opinion that Hippocrates and Galen accurately described the condition. Wohlgemuth found 127 cases reported up to 1923, 79 of which were below the navel and 18 below the semilunar fold of Douglas. No doubt many cases have occurred

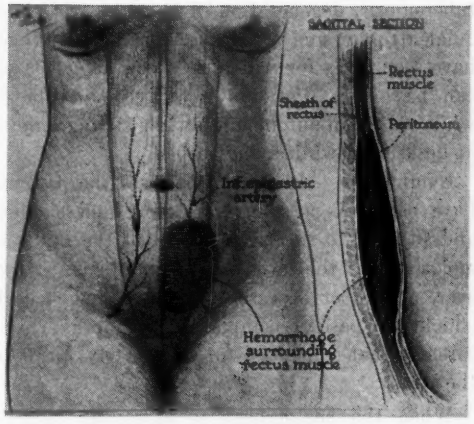
that have never been reported.

Etiology — The most casual study of this subject makes it clear that there is no single specific cause for such hemorrhages. It appears that many conditions, which are the result of internal as well as external influences, act as contributing factors in producing spontaneous bleeding into the muscle following rupture of the deep epigastric artery. Perhaps one of the most important causes is degeneration of blood vessels. While cases are occasionally found in young and healthy persons, in the great majority of instances, the individuals affected have reached or passed middle life. Almost all cases present unmistakable evidence of sclerosis, vascular degeneration, or hypertension.

Some writers attribute the condition to a pendulous abdomen, constant traction upon blood vessels resulting in abnormal changes in the vessel walls. These are unable to withstand the added stress when the muscle itself suddenly contracts or is put to the test by internal pressure as in coughing, sneezing, or, during labor. Other probable causes which may be considered, either with or without a pendulous abdomen, are trauma; a low capillary resistance and a prolonged bleeding time, as proposed by Norgaard; or a chronic productive myositis associated with syphilis, localized arteritis, focal degeneration of muscles and vessels following infections, aneurysmal dilatation, calcification and atheroma of the inferior epigastric arteries that have been included in an article by Giardina.

Numerous cases have been described as occurring during pregnancy, especially in multiparous women; during parturition; and in the immediate postnatal period. Pregnancy is always associated with regeneration and degeneration of muscle fibers — the evolution and involution of muscle tissue. Maxwell has collected 12 spontaneous cases occurring during pregnancy, and 46 attributed to other etiological factors, in not one of which was a correct diagnosis made before operation.

It is also apparent that infection plays an important role in the causation of these hemorrhages into the abdominal parietes, and influenza is the principle infective disease associated with it. The productive factor may be explosive coughing spells and sudden outbursts of sneezing in a disease associated with muscular inanition. In the British Medical Journal, Lieutenant



Colonel Cole gave his postmortem findings in the influenza epidemic at Bramshott in 1918 as follows: "Abscesses were found in some muscles and marked degeneration was repeatedly noted in the rectus abdominis. The muscle looked grey, cooked, and readily flaked three hours after death. Often there was marked hemorrhage, as much as two ounces, into the sheath of the rectus. The muscles were frequently found spontaneously ruptured, as cleanly as though cut through with a knife. The hemorrhage was sometimes present throughout the length of the sheath, though usually only in the lower quarter." Chalier and Vallery describe a case in which there was an infected hematoma of the rectus muscle following typhoid fever and cultures revealed the presence of *staphylococcus aureus hemolyticus*. Others have found a number of cases of infected hematoma but have not given the types of infecting organisms.

Here and there throughout the world authors have described their cases together with many and varied causes, such as the sudden muscular effort connected with some phases of military training which resulted in severe jolting associated with sudden contraction of muscles; the sudden and unaccustomed strain of the muscle in those who try to deceive themselves by crowding all their exercise into one or two days during each month; kicks and blows on the abdomen; a misstep; lifting; strains attendant upon certain forms of manual labor; a hemorrhagic diathesis, in which a study of the blood showed a complete absence of ascorbutic acid; metastatic diseases; alcoholism; and changes which accompany senility. Others have reported cases associated with typhoid and typhus fevers, tetanus,

tuberculosis, degeneration of blood vessels, muscular atrophy, and blood dyscrasias. Still others have seen cases for which no accountable reason can be given. Patients have become afflicted while riding in automobiles, while sitting quietly at desks, and while lying in bed.

Symptoms — In nearly all cases the patients have pain. As a rule it is severe and sudden, and situated to the right or left of the midline, and about at the level of the umbilicus. However, a number of cases have been reported in which the patient complained of more or less soreness that continued for several days prior to the attack.

When the hemorrhage is large the pain is usually severe, and the hematoma may be extremely sensitive to the touch. Nausea and vomiting are of frequent occurrence, and there may be some degree of prostration. The pulse may be accelerated, a slight fever is met with occasionally, and in some cases pallor of the skin and sweating indicate approaching collapse.

The contents vary in size from approximately 64 cc. to 2000 cc. The mass does not change its position, and, if the patient is asked to sit up a little, the enlargement can be felt easily but cannot be moved to either side of the abdominal wall. Another important sign is ecchymosis, which is confined to the area occupied by the extravasated blood. An important sign mentioned by Payne as almost pathognomonic of a lesion in the rectus sheath in the presence of these symptoms is absence of rigidity and tenderness in the abdominal wall adjacent to the tumor, "in contradistinction to what would be expected if there were an intra-abdominal lesion present."

Most of the hemorrhages are unilateral and below the level of the umbilicus. Inasmuch as the epigastric vessels occupy the dorsal surface of the rectus muscle, the greatest amount of bleeding occurs between the muscle and the posterior sheath above the fold of Douglas and between the muscle and the peritoneum below the fold. Numerous cases have been described in which the entire muscle from above the navel to the symphysis pubis has been badly torn by the extravasated blood. In some cases part of the muscle sloughed off in fragments. In others only single strands of muscle were torn.

Finally, the surgeon must not forget that, as a rule, the intensity of clinical manifestations varies with the severity of the hemorrhage. In spite of the absence of rigidity suggested by Payne, a few cases have been reported where rigidity was present and where it was a deciding factor in the diagnosis of an intra-abdominal, inflammatory lesion. This is quite logical when we consider that the location of the hematoma is anterior to the peritoneum and in contact with it. The blood anterior to the peritoneum may produce just as much irritation as when it is posterior to it (as in ectopic pregnancy) and the symptoms in some respects may be quite similar.

CASE REPORT

Mrs. P. W., aged 61, Para eight, entered the Graham Hospital on February 21, 1939, with the preoperative diagnosis of a strangulated left inguinal hernia. Her past history, which may have had some bearing on the present condition, was as follows: In 1934 she had been admitted to the hospital in a thyroid crisis with a decompensated heart. After prolonged preparation, a large goiter, partly retrotracheal, was removed under local anesthesia. During this sickness she lost 84 pounds, which was regained subsequently. In 1938 she was again admitted, with a complete procidentia, which was corrected by a vaginal hysterectomy. Recovery was uneventful. Late in 1938 she had exacerbations of an old hypertrophic arthritis, which compelled her to be very inactive physically and she gained weight up to about 230 pounds.

About a week before her last hospital admission she developed an acute bronchitis and had frequent paroxysms of coughing. Because of her flabby musculature due to obesity and previous illness, these paroxysms caused very much discomfort and abdominal soreness.

On the morning of February 21, 1939, after one of the hard coughing spells, she had a severe soreness in the left lower quadrant, followed shortly by a painful swelling. She was seen by a physician, who though she had developed a hernia and who advised cold applications and rest in the hope that it would return to the abdomen. Her physician saw her again that evening and found the mass larger, irreducible, and more painful than ever, so he sent her to the hospital with the diagnosis of a strangulated or incarcerated left inguinal hernia.

Upon examination at the time of admission, the patient was noted to be quite obese, weighing 212 pounds. She complained of pain in the

lower left portion of the abdomen, which seemed to be aggravated by a severe cough. There had been vomiting on two occasions that afternoon, but no nausea at the time of examination. Her temperature was 100.8°, pulse 88, blood pressure 186/92, hemoglobin 13 grams, red blood count 3,800,000, white blood count 12,250, differential normal, and heart normal. Coarse rales were evident in the lungs. The urinalysis was normal.

Abdominal examination revealed a very large full abdomen with a swelling in the left lower quadrant. This had the appearance and sensation upon palpation of a large incarcerated hernia. The mass was full, rather tense, and so tender that only light palpation was practicable. The surface area was about 21 x 18 cm. with its lower border just above the symphysis pubis.

Because there was no nausea or vomiting at this time, and on account of the severe cough, it was thought best to wait until the following morning before attempting any surgery.

The patient rested quite well that night aside from paroxysms of coughing, and the next morning she had a normal bowel movement. Aside from the tender mass, there were no other abdominal findings, and her condition seemed to be improved. It was then decided to treat the respiratory infection with rest and medication until she became a more suitable risk, and to keep her under close observation in the meantime, in case symptoms suggesting intestinal obstruction arose. No such symptoms occurred and the cough improved readily, but the mass did not change in size although the tenderness did diminish somewhat. No discoloration of the skin was apparent at any time. Her temperature varied from 99.8° to 101.6°.

On the morning of February 27, 1939, six days after admission, the patient's cough had improved to such an extent that exploration of the mass seemed to be justified. Symptoms indicated no intra-abdominal pathology, and there was no sign of obstruction, so an incarcerated hernia seemed the most probable diagnosis. Under ethylene anesthesia an incision was made along the outer border of the left rectus. When the outer sheath was opened, a large hematoma was evacuated. It contained about 250 cc. of clotted blood and when this was removed the rectus muscle was found to be damaged and friable as from pressure necrosis, fully half of the muscle having been destroyed. The hematoma was explored, and the bleeding was found to have come from a large branch of the deep epigastric artery. This was ligated and the anterior rectus sheath was repaired by imbricating it with interrupted ribbon gut.

The patient made an uneventful recovery and was discharged from the hospital on March 9, 1939, with the wound healed and in good condition. When last seen, two months later, she had no symptoms and the abdominal scar was normal.

SUMMARY

1. A case of hemorrhage into the sheath of the left rectus abdominis muscle is reported.

2. Many abnormal bodily conditions have been contributing factors in producing spontaneous hemorrhage into the sheath of the muscle. There is no specific etiologic cause for it.

3. Paroxysms of severe coughing appear to be one of the principle etiological factors.

4. Changes which accompany the middle period of life and thereafter, serve as contributing causes in producing this condition.

5. In comparatively few instances has a correct diagnosis been made.

6. Spontaneous rupture into the rectus abdominis muscle should not be regarded as being of rare occurrence.

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Says Nature to Physic what pity that we
Who ought to be friends should so seldom agree.
With medical legions my humours they chase,
Till pallid resentment appears on my face;
Aperients, astringents, narcotics, combine
To thwart and oppose me in every design;
And by volleys of pills discharg'd at my head
My strength is exhausted, my energy dead.
But Physic should know I am not to be taught
By severe flagellation to do as I ought;
That my faults may be mended by gentle correction
To which science and talents must give the direction.

Nature and Physic, Professional Anecdotes, 1825
Rep. in Can. Med. Assn. Jour., July, 1939.

BONE CARCINOMATOSIS SIMULATING PERNICIOUS ANEMIA

(A Case Report)

B. MARKOWITZ, M.D.
BLOOMINGTON

While one of the most common malignancies, yet carcinoma of the stomach rarely metastasizes to the bone; the figures vary between 2 and 5 percent. (Symmers,[1] Warwick,[2] Kerr & Berger[3]). Its frequency however should not preclude its premortem diagnosis. Metastases to the bone often causes stimulation of the marrow which produces a severe anemia easily misinterpreted as pernicious anemia. (Ellerman[4]). Laurence and Mahoney[5] report one such case of anemia in which they found thrombopenic purpura with marked decrease in platelets, explained on the invasion of the marrow by large numbers of tumor cells. Mallory[6] reports a polypoid carcinoma of the stomach in a patient with pernicious anemia and suggests that pernicious anemia patients should be studied for carcinoma. The clinical impression too may be compatible with a primary anemia. Boyd[7] in differentiating pernicious from other forms of anemia states that one case was extremely suggestive of pernicious anemia even to the extent of presenting peripheral sensory disturbances. Autopsy revealed a carcinoma of the stomach. If the carcinoma is in a so-called silent area of the stomach we can readily appreciate how gastric symptoms will be lacking while the picture of pernicious anemia will stand out prominently. There are however certain factors, as stressed by Jaffe,[8] which speak against this diagnosis and should make one suspicious of bone metastases. If one is alive to the possibility of its presence the diagnosis can more often be made premortem.

Figure 1: Non-ulcerating elevated rounded carcinoma of stomach.

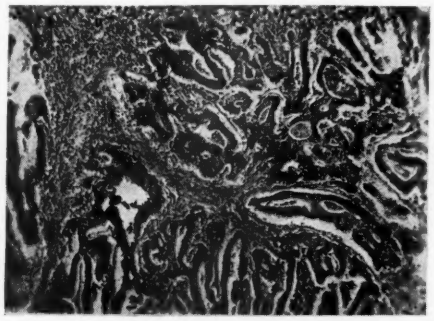

Figure 2: Typical adeno-carcinoma of stomach.

The following case history is quite typical of the marked blood changes similating pernicious anemia together with other factors which speak against it.

This sixty year old white female complained of weakness and pains in her legs of six month's duration. The previous history was irrelevant except that she had been on liver treatment for pernicious anemia. On physical examination the patient did not appear acutely ill but was very pale and very weak. The skin was rather sallow, the mucous membranes pale; the tongue was smooth, the heart and lungs were negative and no masses were felt in the abdomen. The temperature, pulse, respirations and blood pressure were all within normal limits. The red cells numbered 1,010,000, the white cells 10,850, the hemaglobin estimated at 18% (Sahli) giving a color index of 1.1. On differential count there were 66% polymorphonuclears, 26% lymphocytes, 5% myelocytes and 3% eosiniphiles; while counting these 100 white cells, 16 normablasts were encountered. There was moderate disproportion in the red cells with increasing numbers of macrocytes and moderate poikylocytosis. The urine was negative. The Ewald meal yielded no free H C L and 10% total acidity. The blood Wassermann was negative and the metabolic rate was plus two.

The most likely diagnosis was pernicious anemia. The patient was transfused and given liver subcutaneously as well as per mouth. The clinical response however was very poor; nor was there much change in the blood picture after two weeks of treatment. Because of the failure to respond to liver therapy the diagnosis of pernicious anemia was questioned and a low grade infection was considered. Before any more

could be done the patient took a rapid down-hill course and expired three weeks after liver therapy was begun.

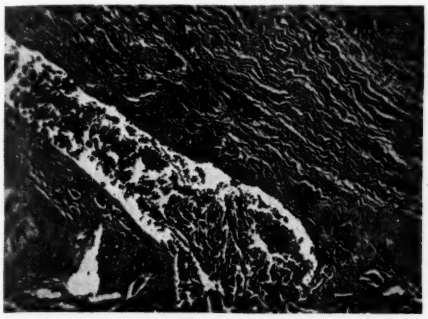

Figure 3: Tumor cells with no definite arrangement in bone marrow.

On post-mortem examination the essential findings were in the stomach and bone marrow. The stomach was of normal size but along the greater curvature, in about mid-portion, was found an elevated, rounded tumor measuring 4½ cmm. in diameter. It was firm in consistency and the mucosa covering it was firmly adherent but intact; there was no evidence of ulceration. The peri-gastric and peri-pancreatic lymph nodes were large, relatively soft and purple gray in color. Microscopic examination of the tumor and adjacent lymph glands revealed the typical picture of an adeno-carcinoma. The bone marrow was bright red in color interspersed with small, purple gray areas. On microscopic examination large groups of tumor cells, with no definite arrangement, were found invading the

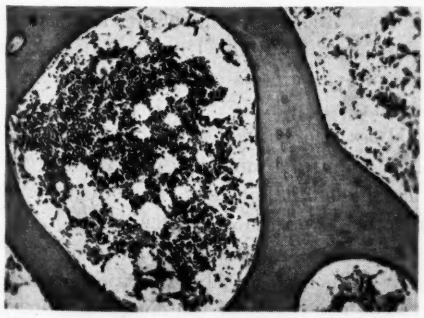

Figure 4: Marrow tissue rich in myelocytic cells. (low power)

bone. In some areas the strands of marrow tissue were rich in myelocytic cells.

The spleen was enlarged to about twice the normal size, weighed 308 grams, was quite firm and the cut surface was bright purple red. Microscopically the splenic pulp showed extensive myeloid metaplasia; many myeloblasts, myelocytes, leucocytes and nucleated red cells. The liver was reddish brown, weighed 1910 grams, and the cut surface presented obscured structural markings. The microscopic section showed many immature blood cells, especially of the myeloid series within the portal capillaries. The remaining organs presented nothing contributory.

COMMENT

The clinical interpretation of this case was confusing. The symptomless primary malignancy of the stomach was not considered because: 1 — located in the greater curvature, without a tumor mass presenting in the lumen, there was no anatomical interference with the emptying of the stomach. 2 — without erosion

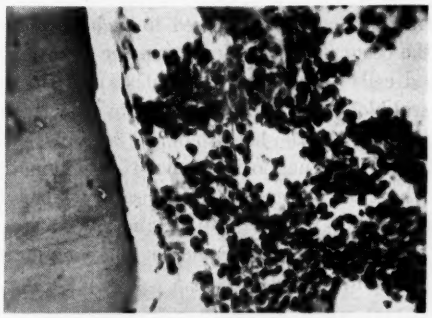

Figure 5: Marrow tissue rich in myelocytic cells. (high power)

of the mucous membrane there was no evidence of blood or irritation of the mucosa to produce gastric symptoms. 3 — even if an X-ray examination had been made it might have been overlooked because of its anatomical arrangement and its location in a so-called silent area.

The blood picture suggesting a primary anemia has often been noted in metastatic carcinoma of the bone. These blood changes consist usually of large numbers of immature cells of both the granular and erythrocyte series in the peripheral blood together with a high color index. The severe anemia of one million red cells and 18% hemaglobin in a very pale appearing individual, not acutely ill, was compatible with the diag-

nosis of pernicious anemia. The large number of myelocytes and normablasts are however not typical of primary anemia and when present should suggest the possibility of bone metastases. In this case there were 5% myelocytes and while counting 100 white cells 16 normablasts were found. This fact, together with the history of previous liver therapy with no apparent benefit, should cause one to question the diagnosis of pernicious anemia and suspect carcinomatosis of the bone marrow. After the patient failed to respond to the liver therapy the diagnosis was finally questioned and this failure to improve on liver confirmed the hematological findings.

CONCLUSION

While certain malignant neoplasms have the tendency to metastasize diffusely to the bone, this type of metastases is relatively rare in carcinoma of the stomach. The diffuse permeation of the bone marrow by the tumor may provoke a severe anemia which closely resembles pernicious anemia. Opposed to such a diagnosis is the occurrence of a large number of immature blood cells (especially normablasts) in the peripheral blood. Failure to respond to liver therapy, together with such hematological findings, should prompt the dnagnosis of bone carcinomatosis. In the case presented, compensatory blood formation appeared in the liver and spleen because of the extensive involvement of the bone marrow by the tumor tissue.

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CLINICAL ASPECTS OF VITAMIN DEFICIENCIES IN OPHTHALMOLOGY

PETER C. KRONFELD, M. D.

CHICAGO

I may owe the privilege of addressing you on the clinical aspects of vitamin deficiencies in ophthalmology to the fact that for six years I was connected with the Peiping Union Medical College in Peking, China, where I had ample opportunities to study practically all types of vitamin deficiency in very severe forms. The tremendous size of the vitamin problem in China may make me unduly underrate the significance of vitamin deficiencies in this country. If in the following I should make this mistake, I wish to apologize for it beforehand.

The unbiased observer of the situation as it prevails in the United States with regard to the use of vitamins is bound to recognize the effect of two, what I would call distorting, factors, namely the effect of intensive propaganda on the part of the vitamin manufacturers directed at lay people as well as at doctors, and also the injudicious enthusiasm with which doctors as well as lay people welcome any new therapeutic principle. Let us try to reduce to a minimum the influences of these two factors in our discussion today.

The wealth of published evidence, clinical observations, theories, and wishful thinking which concern practical clinical aspects of vitamin deficiencies in ophthalmology may be grouped into three categories in the form of answers to the following three questions:

1. Does the ophthalmologist in private or clinical practice in the United States see cases of frank avitaminosis or hypovitaminosis of the particular vitamin?

2. Does the particular avitaminosis produce ocular manifestations by the recognition of which the ophthalmologist can contribute to the diagnosis of vitamin deficiency?

3. Are there ocular conditions which are beneficially influenced by administration of amounts of vitamin in excess of the normal requirements (indications for supplementary vitamin administration)?

From the Illinois Charitable Eye and Ear Infirmary, Chicago, Ill.

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VITAMIN A

Incidence of avitaminosis and hypovitaminosis. Frank avitaminosis A is very rarely seen by the ophthalmologist practicing in the U.S.A. In general, the intake of vitamin A of the population of the U.S.A. is ample so that deficiencies are likely to occur only in severe prolonged disturbances of absorption from the gastrointestinal tract, in severe liver disorders in which the conversion of carotene into vitamin A is impaired, in conditions of increased consumption (hyperthyroidism) or of increased expenditure (pregnancy, lactation) of vitamin A. Even in the pediatric wards of the hospitals for the indigent, keratomalacia has become extremely rare.

The incidence of hypovitaminosis A among the population of the U.S.A. is a controversial subject. There is ample physiological and clinical evidence to the effect that the optimal intake of vitamin A is very much greater than the minimum which barely prevents the occurrence of definite deficiency symptoms. Especially in the children of the poorer classes who receive only limited amounts of milk, cream, butter and green vegetables hypovitaminosis A is likely to be present especially if there is a disorder of absorption which aggravates the condition. In eye clinics hypovitaminosis A has therefore to be reckoned with and probably is present in a considerable percentage of children with phlyctenular disease.

According to a number of reports from European countries[1], the course of inflammatory corneal diseases on the order of phlyctenular disease, herpes simplex, punctate superficial keratitis in individuals with hypovitaminosis A is influenced beneficially by correction of the latter. This is understandable since a certain vitamin A content is apparently necessary for the functional integrity of the epithelial cells and therefore also for their resistance to noxious agents of all kinds. In chronic or recurrent superficial inflammatory diseases of the cornea and conjunctiva it would therefore seem good practice to keep hypovitaminosis A in mind as a possible debilitating or perhaps predisposing factor.

Ocular manifestations of avitaminosis A. An ocular condition supposedly related to hypovitaminosis A is the form of asthenopia associated with photophobia which occurs in people exposed to excessive amounts of sunlight. Cordes and Harrington[2] reported this syndrom to occur fairly frequently in California and to respond well to daily doses of 30,000 units of vitamin A or carotene. In some of these patients a hypovitaminosis A may have been present, since there was a complaint of "night blindness" in 22% and a history of intentional dietary restrictions in a still higher percentage. This form of asthenopia may be characteristic of sunny climates. Here in the Middle West it appears to be rare. It may, however, occur in certain occupations such as, for instance, the personnel of gasoline stations and should then be treated according to the recommendations of Cordes and Harrington.

From the very characteristic and conspicuous ocular manifestations of avitaminosis A the names "antixerophthalmic vitamin" or "xerophthol" have been derived. Objectively, a diffuse mild xerosis of the conjunctival and corneal epithelium, with or without circumscribed accentuation in the form of Bitot's spots, but always associated with reduced production of tears is the earliest and very characteristic ocular change[3]. In North China the ocular manifestations are usually preceded by skin changes in the form of follicular hyperkeratosis[4].

Because of the fact that vitamin A is a component of the visual purple the faculty of dark adaptation is intimately related to the vitamin A metabolism of the body. On the basis of adaptometric studies the diagnosis of hypovitaminosis A has been made by pediatricians and internists in this country. The very exact work on the dark adaptation of normal individuals who for varying periods reduced their daily vitamin A intake to less than 300 units which was undertaken in several American institutions[5, 6, 7] during the last two years has brought out clearly the multiplicity of factors involved in adaptometric work. The test is entirely subjective and requires a much keener power of observation than is necessary for perimetric work. It seems doubtful whether all the individuals who were tested by internists and pediatricians possessed sufficient power of observation to make their statements — some of them were children — appear reliable. Furthermore, in order to obtain reproducible results a fairly complicated apparatus allowing accurate control of fixation and of other factors is absolutely necessary.

To me it seems rather questionable whether the ophthalmologist by means of a crude adaptometer will be able to definitely draw a borderline between normal and slightly impaired dark adaptation in the average patient. Doubtlessly the severer degrees of night blindness as they occur in marked vitamin A deficiency or in retinotapetal degeneration can be diagnosed very readily with very simple adaptometers on the order of the one designed by Birch-Hirschfeld, but unless the conditions are really ideal for accurate adaptometry the diagnosis of mild hypovitaminosis should in my opinion only be made if the results of the dark adaptation test are corroborated by other findings, like hyperkeratosis of the skin or a low vitamin A content of the serum.

There is evidence to the effect that the milder degrees of night blindness are associated with a contraction of the visual field for yellow only[8]. If the ophthalmologist has at his disposal a perimeter which permits perimetry under standardized and accurately reproducible conditions, the determination of the isopter for a yellow target of 3-5/333 may be a valuable substitute for adaptometric studies.

Indications for supplementary vitamin administration. The ophthalmological literature abounds in reports on ocular conditions which have been beneficially influenced by oral or local administration of vitamin A in patients without definite signs of avitaminosis or hypovitaminosis A. This practice has by some authors been criticized as "indiscriminate" use of vitamins. In principle, however, no serious objection can be raised to such a procedure. The doses of vitamin A used were certainly harmless so that their therapeutic effect could only have been nil or beneficial.

This brings us to the much debated subject of the rationale of local use of vitamin A. Its introduction into the treatment of wounds by Löhr in 1937 was at first greeted very enthusiastically. In a recent resume on "The Pharmacology and Therapeutics of Vitamin A" Clausen[9] sums up the situation by saying that "the experimental work done throws doubt on any specific action of the vitamins contained in the preparations applied locally." In order to ascertain the value of such therapy applied to the eye de Roetth[10] studied the effect of local application of the vitamin on experimental keratomalacia in rats. His conclusions were: "Oils containing vitamin A have a definite systemic, not a local action when applied to the eye. It is not known, however, whether it is resorbed by the surface of the eye or whether it reaches the stomach through the nasal duct." In normal rats in whom equal portions of corneal epithelium were scraped off "there was no difference in the time necessary for epithelization when the right eyes were treated with cod liver oil and the left eyes with liquid petrolatum. These experiments do not confirm the idea that vitamin A acts locally to promote epithelization." Does this mean that the local application of vitamin A preparations in inflammatory or traumatic lesions involving the anterior surface of the eye is altogether pointless? That conclusion would in my opinion go a little too far.

From the purely clinical point of view the experiences with local application of vitamin A in various inflammatory, traumatic or degenerative diseases of the conjunctiva and cornea have been, to say the least, encouraging. The excellent results reported by Russian authors[11] may be partly due to a prevalence of a marked hypovitaminosis A among their patients. This condition did probably not apply to the series of cases in Great Britian, Germany, Italy and Hungary who were subjected to local vitamin A treatment. Stevenson[12] found locally applied cod liver oil to possess "an undoubted and rather surprising efficacy as a healing agent." De Grosz[13] sums up his experiences by saying that local application of vitamin A "possesses a large field in ocular therapeutics; it represents an outstanding therapeutic agent, especially for injuries and trophic conditions of the cornea.

My feeling in the matter is that the local application of vitamin A has proved to be a very effective way of administering the vitamin. In the severe avitaminoses of Chinese children in which the absorption from the intestines was often markedly impaired by a chronic gastroenteritis, I found the local application of cod liver oil ointments to be most beneficial and a true "sight-saver" when we had run out of vitamin A preparations suitable for intramuscular injections. In individuals with ample vitamin A intake in their natural food the local application of vitamin A ointments may further increase the vitamin content of the ocular tissues which by a local disease may have become de-

ficient and thus make them independent of the vitamin supply through the blood stream. The possibility thus still remains that in severe burns or necrotizing inflammatory diseases the local application of vitamin A is of some value.

VITAMIN B_1

Incidence of avitaminosis and hypovitaminosis. The picture of the marked avitaminosis B_1 with or without signs of deficiency of the B_2 complex, is seen fairly commonly by the ophthalmologist in the form of the so-called toxic amblyopia due to alcoholism. While a direct toxic effect of the alcohol upon the optic nerve probably does play a part in these cases, the B_1 deficiency which is the result of a reduced intake plus a very much impaired absorption due to the chronic atrophic gastroenteritis characteristic of alcoholism, is the most important factor in the pathogenesis of this disease. The ophthalmologist is usually the first to see these patients and is thus enabled to materially alter the course of the ocular as well as the systemic disease.

Since it is a generally recognized fact that in the U.S.A., Western and Central Europe the B_1 intake of the large majority of the population just about covers the ordinary metabolic requirements hypovitaminosis B_1 is bound to occur in any clinical material.

Ocular manifestations. The ophthalmologist can hardly make a contribution toward the diagnosis of this hypovitaminosis since demonstrable optic nerve lesions apparently occur only in deficiencies of great severity and long duration. In the pure form of the deficiency which is known under the name of tropical or oriental dietary retrobulbar neuritis[14] and which does not occur in the U.S.A. the main ocular symptom is a bilateral central scotoma and not the centrocoecal scotoma which is so characteristic of the B_1 deficiency associated with alcoholism. In the Philippines I have seen B_1 deficiency during pregnancy or shortly after delivery manifest itself by an acute neuritis of the ophthalmic branch of the fifth nerve, leading to an acute and very painful keratitis.

Indications for supplementary vitamin administration. The assumption that the metabolic requirements of traumatized or inflamed nerve tissue are greater than those of normal nerve tissue has led to the use of vitamin B_1 in the treatment of various inflammatory, metabolic and traumatic diseases of the nervous system. The neurologist would be the proper authority to report to you on the results of this treatment, but even the ophthalmologist is bound to recognize the very beneficial effect of large doses of vitamin B_1 in certain neurological diseases. The best example in ophthalmology is herpes zoster in which I have seen it to dramatically influence the pain. To what extent the vitamin therapy tends to reduce the damage to ocular tissues caused by the zoster neuritis is difficult to say since so much of this process takes place in a part of the eye not directly accessible to clinical examination. It is my feeling that the new and apparently very successful treatment of herpes zoster with parenteral injections of diphtheria antitoxin (5000 units) should not supplant the vitamin therapy but rather be combined with the latter.

As far as the ocular manifestations are concerned the various avitaminoses caused by lack of one or several members of the B group present themselves either under the picture of the B_1 deficiency which we have just discussed or under the picture of ariboflavinosis which, from the standpoint of practical ophthalmology in this country, is at the moment probably the most important vitamin deficiency.

RIBOFLAVIN

(vitamin B_2 in the strict sense or vitamin G)

Incidence of avitaminosis and hypovitaminosis. Ariboflavinosis, characterized by cheilosis, glossitis, and seborrhea, is a common disease in all districts of the U.S.A. where pellagra is prevalent. According to Sydenstricker and associates[15] it possibly is "the most prevalent, apparently uncomplicated avitaminosis; it is possible also that it is more easily recognized than others on acount of the specific lesions of the eye which occur early in the period of deficiency."

Ocular manifestations. To the ophthalmologist ariboflavinosis usually presents itself under a picture that until recently has been called acne rosacea, characterized by flushing, teleangiectasia, hypertrophy and pustules of the skin of the face associated by a chronic or recurrent keratoconjunctivitis of which excessive

superficial vascularization with corneal infiltrations and ulcers are the outstanding manifestations.[16] The ophthalmologist is in a position to recognize the earliest stages of ariboflavinosis and thereby to make an important contribution to the early diagnosis of the disease. Observations on animals in which ariboflavinosis has been produced experimentally and in man make it certain that invasion of the cornea by newformed vessels which are derived from the normal superficial limbic plexus is the earliest symptom of riboflavin deficiency[17, 18]. To what extent this invasion can be diagnosed in its beginning depends somewhat upon the extent to which the anatomy of the limbic vessels has been altered by previous corneal diseases. Accurate knowledge of the slitlamp picture of the normal and diseased limbus thus becomes a necessary requisite for the early detection of ariboflavinosis. The vascular invasion is usually associated with slight congestion of the normal limbic vessels, photophobia and itching. Johnson[18] describes the general pattern of the vascularization due to ariboflavinosis as resembling a large unleafed elm branch. Excellent diagrams of these vessels can be found in the article by Sydenstricker and associates[15], who proposed the appropriate name of dietary keratitis for the disease heretofore called acne rosacea keratitis.

In the classical form of ocular ariboflavinosis dramatic improvements and cures have been obtained by administration of the lacking vitamin by mouth or parenteral injection. A necessary condition for the proper utilization of orally administered riboflavin is a fairly acid reaction of the stomach contents which, if one does not care to subject the patient to a gastric analysis, can be brought about conveniently by the administration of small amounts of dilute hydrochloric acid before the ingestion of the riboflavin.

Indications for supplementary vitamin administration. Since the principal action of riboflavin is to act as a catalyzer in the carbohydrate and amino-acid metabolism and since this action is of much greater significance in avascular than in vascular tissues, it is to be expected that riboflavin will prove beneficial in corneal diseases not due to a systemic ariboflavinosis. Up to this time no striking results in such diseases have been reported.

VITAMIN C

Incidence of avitaminosis and hypovitaminosis. Severe avitaminosis C is practically nonexistent among the patients seen by the ophthalmologists practicing in the U.S.A. Using as criterion of hypovitaminosis C the amount of vitamin C necessary for saturation of the body one finds that most of the elderly individuals who are treated by ophthalmologists for senile cataract or glaucoma are in a state of hypovitaminosis C. Some authors[19] state that this hypovitaminosis is more marked in cataract patients than in individuals of the same age but without cataracts, whereas other authors have failed to find such a difference. Ascorbic acid has been shown to play a very important part in the formation of intraocular fluid[20] and in the metabolism of the lens[19]. The discussion of these very interesting relationships would probably be considered a transgression by the program committee and is therefore omitted. Of the practical applications of this work I would like to mention the use of ascorbic acid in large doses by mouth or parenterally prior to intraocular operations upon elderly individuals. The rationale of this therapy is the interpretation of postoperative hemorrhages as manifestations of a hypovitaminosis C. Actually Urbanek and Albrecht[21] and others have reported that postoperative hemorrhages into the anterior chamber do not occur in patients saturated with vitamin C by daily doses of 300 mg. ascorbic before and during the first week following the operation. Since the procedure is simple and free of any disadvantages it should be practiced as an additional safety measure before and after intraocular operations.

Large doses of vitamin C have been used extensively and with very divergent interpretations of its effect in the conservative treatment of senile cataract. My own feeling is that even by very close observation of cataract patients it is very difficult to recognize with certainty the effect of any conservative therapeutic procedure. The metabolism of the cataractous lens has been shown to be faulty and defective in so many respects that flooding of the eye with ascorbic acid alone can not be expected to change the course of senile cataract. I do not, however, want to imply that I consider the conservative

treatment of senile cataract as an absolutely hopeless enterprise. On the contrary, I feel that especially the study of vitamines in relation to lens metabolism has shed a great deal of light on the various metabolic requirements of the lens and has thus shown us what food substances, enzymes and catalyzers the lens requires for functional integrity. Only through such studies can one hope of ever being able to replenish the food stores of the cataractous lens and thus to rectify its metabolism.

Ocular manifestations. Avitaminosis or hypovitaminosis C does not produce any eye symptoms of practical diagnostic value.

Indications for supplementary vitamin administration. Vitamin C has proved to be of therapeutic value as a styptic in a number of diseases characterized by a tendency to capillary hemorrhages not related or due to hypovitaminosis C. "Vitamin C seems to tighten the capillary walls," says W. Stepp in his monograph on vitamines. After parenteral administration of large doses of vitamin C the blood platelet count is usually found to go up and the coagulation time shortened due to an activation of thrombin. On the basis of these facts one is tempted to try vitamin C for the symptomatic treatment of ocular conditions in which intraocular hemorrhages occur spontaneously, in addition to the application of all known measures directed against the etiology of the disease. I am thinking here of the tuberculous form of retinal periphlebitis[22], without doubt one of the most unpleasant diseases from the standpoint of treatment since there always is the possibility of correlating a new hemorrhage with some therapeutic step taken immediately prior to the accident. From the few cases of this disease which I, in addition to very careful tuberculin treatment, have treated with vitamin C, I have received the impression that the latter was of definite value. Whether the course of diabetic retinitis is in any way influenced by vitamin C I am not able to say with certainty at this time. I have, at present, a number of patients under observation who in addition to a very careful diabetic regime are kept on large doses of vitamin C.

VITAMIN D

Incidence of avitaminosis and hypovitaminosis. The ophthalmologist practicing in the U.S.A. rarely comes in contact with cases of avitaminosis D, rickets in children and osteomalacia in the adult. Even in pediatric wards of hospitals for the poor rickets has become an uncommon disease.

Ocular manifestations. The only ocular manifestation of avitaminosis D is the development of bilateral cataracts of the zonular type which, in rickets as well as in osteomalacia[23] is an expression of the profound and long-lasting disturbance of the calcium metabolism caused by vitamin D deficiency. The cataract is a late development and therefore of no practical diagnostic importance.

Indications for supplementary vitamin administration. A. A. Knapp[24] claims "that a disturbance in the vitamin D-calcium-phosphorus metabolism is concerned in the etiology of myopia. In the presence of a calcium imbalance, there may be a weakening of the fibrous tissue which may give rise to myopia. Once a condition of progressive myopia has been established treatment with the vitamin D complex is indicated. The myopic eyes that respond to this therapy may undergo an actual shrinkage of the globe." Although I have not had much faith in this concept I have carried out Dr. Knapp's therapeutic program in a number of youths with progressive myopia. There has been no statistically demonstrable difference in the amount of progression of myopia between these patients and controls who received no vitamin D in addition to their regular diet. In the absence of any more promising "treatment" of progressive myopia in adolescence the vitamin D therapy may deserve a trial.

VITAMIN K

Incidence of avitaminosis and hypovitaminosis. The human body receives its supply to vitamin K party from the green leafy vegetables contained in food and partly through the action of the normal bacterial flora of the intestines which synthesize and store in their bodies large amounts of the vitamin. For its absorption from the intestines the presence of bile is absolutely necessary. Vitamin K deficiency therefore occurs in all forms of obstructive jaundice. Vitamin K is necessary for the formation of prothrombin in the liver; the main deficiency symptom is therefore delayed coagulation of the blood which constituted a serious problem to the

surgeon who had to perform major surgical operations on patients with obstructive surgery. The discovery of vitamin K and the availability of the market of very effective vitamin K preparations — with or without bile salts — has brought about a very dramatic change in the treatment of patients with obstructive jaundice.

Hypovitaminosis K which occasionally occurs in certain forms of mild gastroenteritis or colitis can be diagnosed by determining the prothrombin content of the blood, a fairly simple procedure which is carried out in any clinical laboratory. In patients who give a history of chronic digestive disturbances it may be wise to have the prothrombin content of the blood determined before any intraocular operation is performed.

Indications for supplementary vitamin administration. Whether or not the administration of vitamin K in excess of the normal requirements shortens the normal blood coagulation time is not definitely known. In patients with unexplained post-operative hemorrhages but normal coagulation time vitamin K has been used by a number of ophthalmologists with apparently beneficial results. Burch and Meade[25] have reported a favorable response to vitamin K therapy in a case of retinal hemorrhages of unknown cause.

SUMMARY

The vitamines A, B_1, riboflavin, C, D and K play important parts in the normal functioning of the ocular tissues. Avitaminoses are associated with characteristic ocular manifestations some of which occur at an early stage and are therefore of diagnostic significance. In a number of diseases the administration of vitamines in excess of the normal requirements has proved to be of great therapeutic value.

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CLINICAL ASPECTS OF VITAMIN DEFICIENCY IN THE EAR, NOSE AND THROAT

LINDEN WALLNER, M.D.,
CHICAGO

The manifestations of true deficiency diseases are so rare in the ear, nose and throat that on first thought vitamins may not be considered important. Specialists are often criticized for a too narrow outlook. It is well to review the field of avitaminosis, to see if there are diseases ordinarily ascribed to other causes that may be due, at least in part, to insufficient vitamin intake.

VITAMIN A.

This has been called the anti-infective vitamin, a term given by Mellanby in 1926 because of the chronically infected noses and frequent upper respiratory infections he found in infants on *A* deficient diets. Widespread advertising has furthered this anti-infective idea among the public. Vitamin A preparations are given routinely to the personnel of large companies for the prevention of colds.

The experimental evidence is found in animals fed deficient diets and from clinical studies of its use on large control groups.

In an important study in 1925 Wolbach[2] and Howe fed rats Vitamin *A* deficient diets. The

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epithelium of the upper respiratory tract and sinuses underwent a metaplasia from the normal ciliated columnar to a keratinized, stratified squamous type, followed by infection. This has been verified[3,4] by many other workers and all[1] find animals on A deficient diets develop infections of the upper respiratory tract.

There have been numerous clinical[5,6,7,8] studies of the effect of vitamin A and colds.

Some merely give a vitamin concentrate, and the number of colds was recorded, compared with the number the subject thought he had the year before. Others compare the results with a similar group as controls.

One of the best of these studies was by Jeghers[9] on 162 medical students, 50 or 36% were found to have sub-normal light adaption and their calculated vitamin intake was ½ of the others. The incidence of colds was about the same, though the duration was longer in the vitamin deficient group. Others[10] gave an excess of vitamin A to groups already known to be receiving an adequate intake. There was no lowered incidence of colds but a suggested shortening of duration and lessened severity.

No systemic[11] lack of immunity or anti-body production has been demonstrated in vitamin A deficiencies.

The present status of vitamin A in the ear, nose and throat may be summarized:

1. Vitamin A is necessary to the health of the mucus membrane of the upper respiratory tract. When deficient, there may be lowered local resistance to bacterial infection.
2. The epithelial metaplasia in experimental animals is a late result, it is *not* seen in humans, with rare[12] exceptions. The stratified squamous epithelium in atrophic rhinitis has been compared to the metaplasia of animals. This disease has long been thought to have a dietary etiology. Its rarity, even in clinics today, suggests better feeding, especially of infants.
3. There is no special characteristic of infections in the ear, nose and throat that identify it as due to lack of vitamin A. Neither do we have a simple method of determining the presence of A deficiency. A calculation from the diet may help. Blood level determinations or dark adaptation tests are not practical for routine office or clinic use.
4. Insufficient intake of vitamin A may be one factor to be considered, along with allergy, infection, humidity and many others in persons subject to frequent colds.
5. The administration of vitamin A to persons already receiving an adequate intake in the diet does not lessen susceptibility to colds. It may shorten their duration or lessen their severity.
6. The term anti-infective vitamin is not justified.

VITAMIN B.

Including B_1 thiamin chloride, and other factors of the B complex.

One of the known changes in B deficiencies is the nerve degeneration, specifically de-mylenization. Possible applications that suggest themselves in otolaryngology are in diseases of the 8th nerve, in the cochlear division, deafness; and the vestibular portion, vertigo; as well as the many forms of neuralgia about the head.

Eighth Nerve. It has been shown that experimental animals (chicks, rats[13, 14]) on diets lacking in various factors of the B complex, undergo degeneration, demylenization of both the cochlear and vestibular nerve.

Selfridge has written extensively on both the experimental and clinical aspects of vitamin B complex deficiency as a factor in the cause of nerve deafness. He grants it is often a senile change, but feels that the degeneration of age is caused or accentuated by vitamin deficiency. He offers a large series of cases, with audiograms before and after treatment, showing definite, though not marked, improvement in hearing in many after administration of various factors of the B complex. There is nothing distinctive in the audiograms to indicate the cases as different from the usual primary nerve deafness. Other[15] observers have reported similar studies with poorer results.

I have been interested in a series of nerve deafness cases treated by a colleague with vitamin B, in large doses with entirely negative results. There was no improvement of hearing in the cases of nerve deafness which I have treated with B_1.

It is difficult to believe that mild, sub-clinical avitaminosis is a real factor in nerve deafness,

when we remember defective hearing is not a symptom of the definite B deficiency diseases, beri-beri or pellagra. Tinnitus[16, 17] and vertigo have been described in pellagra however.

The changes observed in animals in the vestibular nerve have suggested the use of B_1 and B complex in Menieres syndrome. Harris[17] and Moore noted peculiar dietary habits in persons with Menieres disease. Large doses of thiamin were ineffective. Then they tried nicotinic acid with negative results. Used together, 250 milligrams of nicotinic acid and 20 milligrams of thiamin per day, 17 of 20 cases were entirely relieved, 3 improved. In 10 the tinnitus disappeared and was improved in the others. In 12 the hearing was improved.

Atkinson[18] divides Menieres cases into those due to allergy and those due to vascular spasm. The latter are mostly in older individuals and respond to B_1 and nicotinic acid. He feels the improvement is due to a vaso-dilator action, rather than the correction of vitamin deficiency.

Cases of pain about the head, over the sinuses, nose, face and throat without apparent disease are frequent in oto-laryngology. These have been most difficult to handle, they are often labeled as "neurotics." The use[19] of Thiamine Chloride has been a distinct advance and worth a trial before alcohol injections or surgery. Fifty milligrams of Thiamine Chloride subcutaneously daily for 10 days is an adequate trial. The relief may be due to correcting of a deficiency or some "non-specific" action of B_1 as some feel.[19]

The lesions in the mouth, tongue and pharynx in pellagra and those of the lips, cheilitis, in riboflavin deficiency should be mentioned.

To summarize B_1 and B complex in oto-laryngology:

1. There is no disease syndrome in the ear, nose, or throat characteristic of B deficiency.
2. Good results have been reported in its use in case of nerve deafness and Menieres disease. Its use empiracally in these cases may be justified.
3. Its use in the troublesome and baffling neuralgias about the sinuses, throat and head is justified. It is simple, easily given, relieves many of these that otherwise would need alcohol injections or more drastic surgery.

VITAMIN C.

Ascorbic or Cevitamic acid. The classic manifestations of a lack of vitamin C is scurvy. This disease will not be discussed here, though it may be necessary to recognize or differentiate its oral manifestations in laryngology.

Hemorrhage from the nose may also be a symptom even in so called sub-clinical scurvy.

With the exception of scurvy, there are no characteristic conditions of the ear, nose or throat typical of vitamin C deficiency.

VITAMIN D.

Vitamin D is known to control bone metabolism by the utilization of phosphorus and calcium. The disease in oto-laryngology where bone metabolism is important is otosclerosis. Lesions in the labyrinth similar to otosclerosis have been reported[20] in experimental animals on a diet deficient in vitamin A and D.

Clinically there is no evidence that a lack of vitamin D is a causative factor in otosclerosis or that its administration influences its progress.

VITAMIN K.

The beneficial effect of vitamin K in hemorrhage is in cases due to lack of prothrombin, usually associated with a lack of bile salts. Surgery of the ear, nose or throat is not often necessary in jaundiced individuals. A lack of prothrombin has been shown after massive hemorrhage. Further studies may show the value of vitamin K in hemorrhage such as severe tonsillar or nasal.

SUMMARY AND CONCLUSIONS

1. It is assumed that an adequate vitamin intake is necessary for general bodily welfare and therefore to the normal state of the ear, nose and throat.
2. We must be critical of adapting to humans the findings in experimental animals fed on diets deficient in the various vitamins.
3. Diseases of the ear, nose and throat characteristic of avitaminosis are rare. However, it may be a contributing factor in a number of conditions ordinarily considered due to another etiology.
4. The use of vitamin preparations is worth trial clinically in a number of ear, nose and throat

conditions, even though no definite lack of intake is shown.

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MANAGEMENT OF PERIPHERAL VASCULAR DISEASE

GEZA DE TAKATS, M. D.

CHICAGO

The treatment of peripheral vascular disease rests on adequate diagnosis. A simple classification of the various disorders of peripheral circulation should always be kept in mind, as it will clarify the indications for the diverse forms of treatment. One must ask one's self whether or not a vascular occlusion is acute or chronic, organic or functional, inflammatory or degenerative, spastic or paralytic. Most of the bizarre and combined types of peripheral vascular disease can be brought down to a few common denominators (table 1). It is obvious that no single case

TABLE 1 — Classification of Peripheral Vascular Disease

Organic		Functional	
Acute thrombosis	Chronic congenital traumatic inflammatory degenerative	Vasoparalytic	Vasospastic neurogenic endocrine metabolic toxic
embolism			

of vascular disease is purely organic or purely functional. It is the purpose of an examination of the peripheral vascular state to determine the various elements in the individual case and to estimate for the sake of prognosis and type of treatment, the reserve capacity of the vascular bed, or, expressed differently, the extent of the organic damage.

The objectives of treatment may be readily summarized under three headings (table 2).

TABLE 2 — Objectives of Treatment in Peripheral Vascular Disease

1. To improve collateral circulation.
2. To alleviate pain.
3. To remove non-viable parts at the optimal time and level.

These can be accomplished by physical therapy, by drugs, and by surgical procedures (table 3).

TABLE 3 — Methods of Treatment for Peripheral Vascular Disease

Physical	Medical	Surgical
Heat	Vaccines	Sympathectomy
Hot and cold bath	Hypertonic solutions	Paravertebral block
Postural exercises	Alcohol	Peripheral nerve block
Massage	Theobromine	Amputation
Diathermy	Papaverine	
Short wave	Nitrites	
Iontophoresis	Choline derivatives	
Suction and pressure	Iodides	
Venous Hyperemia		

From the Department of Surgery, University of Illinois, College of Medicine. Address given at Post Graduate Conference of the Illinois State Medical Society, La Salle, Illinois, October 3, 1940.

An intelligent coordination of these procedures will yield the best results. As a recent monograph stated, "Because one is a physiotherapist, is no excuse for the employment of physical measures exclusively in the treatment of peripheral vascular disorders. Nor is there any justification for the indiscriminate performance of ganglionectomy, because one is a neurosurgeon." To this I may add that some of our internists are also inclined to push fluids and drugs through all available channels and ignore other forms of therapeutic approach.

Coordination of these various measures may be accomplished by men familiar with peripheral vascular disease, but chiefly by the family physician, who can see to it that specialists do not run away on one of their individual hobbies. There are, of course, several ways of getting well, and the social and economic status of the patient is one of the important factors in selecting types of treatment.

Physical Therapy in Peripheral Vascular Disease

Heat in all forms is useful, but the cheapest, safest, and most effective is the continuous or intermittent use of a "baker." If the patient is bedridden, heat should be applied continuously. If the patient is ambulatory, it is easy to utilize the night for a prolonged use of the baker. The temperature is either under thermostatic control or can be adequately regulated by the number and intensity of electric bulbs. The temperature within the baker must not be so high as to require more blood and more oxygen than the narrow or occluded vascular channels can deliver. Most patients with arterial occlusions are comfortable between 85 and 90 degrees F., but occasionally the optimal temperature is lower than that; as low as 75 degrees F. With the lower temperatures, the rate of oxidation in the tissues is slower and there will ensue a diminished requirement for oxygen. If the patient is complaining of pain under the heat cradle, it signifies that he is getting too much heat. This is just as harmful as if his feet were too cold. While heat is an active vasodilator, it must be remembered that in the presence of a greatly diminished flow of blood vasodilatation will increase stasis and require too much oxygen. In case of heat-intolerance, it is best to apply heat to the root of the limb. Generally speaking, it is safest to wrap the affected limb in cotton and move the cradle up toward the root of the limb.

Alternate hot and cold baths are frequently used to "tone up the blood vessels." In the experience of our group their value is rather questionable in peripheral vascular disorders. They tire the patient, macerate his skin, cannot be used in the presence of gangrene, and may even aggravate vasoconstriction. To my mind even mild vasodilatation is far more desirable than alternate thermal insults, which do not provide rest for the blood vessels. Better than that is a warm soak for 15 or 20 minutes, preferably with a mild antiseptic, such as a 1:8000 solution of potassium permanganate. The latter controls the often coexisting ringworm infection, which may be the starting point of serious infection, gangrene and loss of digits or limb. Warm sitz-baths at night are useful.

Postural exercises have a sound foundation, and oscillometric curves illustrate the increasing pulse volume in the dependent position. They may be done actively by the patient or passively by an oscillating bed. The patient can also elevate the leg with the help of a pulley, which saves a lot of oxygen that would otherwise go to nourish the contracting muscle. This passive elevation can be combined with intermittent venous hyperemia, which will be discussed later.

Massage is a time-honored method to improve circulation. In the absence of venous or recent arterial thromboses and when the skin is intact, mild massage may overcome vascular spasms and produce a reactive hyperemia. As will be discussed below, we have far more efficient methods to produce reactive hyperemia. In our clinic, perhaps for economic reasons, we have made little use of massage.

Diathermy treatments. — To heat up the lower extremity, electrodes are placed on the abdomen and the lower back. For the treatment of the upper extremity, the electrodes are placed on the side of neck and the upper dorsal spine. Thus not only is the blood being heated as it passes through the root of the limb but a reflex vasodilatation is produced, which is mediated through the sympathetics, as it is absent in sympathectomized patients. We have not placed electrodes on affected limbs, as they are increasingly sensitive to burns. In our experience the application of hot water bottles to the root of a limb, or heating the entire body with a large cradle, is almost as effective and much cheaper. These reflex heat treatments are especially valu-

able in relieving vasospastic phenomena, so that they can be advantageously combined with the newer mechanical forms of stretching the vessels, such as suction and pressure.

Short wave therapy, judging from reports, should be even more effective than conventional diathermy. Again, applying it to the root of the limb, both the vessels and the vasomotor supply are affected. Further study is necessary to establish its value and indications in the treatment of peripheral vascular disease.

Iontophoresis. Vasodilators such as histamine, acetylcholin, and mecholyl have been introduced through the intact skin by placing them on the positive pole and carrying them into the tissues by the galvanic current. We found in unpublished observations that the skin often developed a refractory state, with increased pigmentation and thickening and that the liberation of vasodilator substances from the patients own tissues was a far simpler and more economical method. The use of accumulating tissue products will be discussed under the heading of intermittent venous hyperemia. The use of sodium chloride, however, by iontophoresis has given encouraging results in scleroderma and thrombophlebitic indurations.

Passive Suction and Pressure Exercise

A physical method of dilating collateral arterial pathways of an extremity, whose major arterial pathways had been occluded by trauma or disease, has been called passive vascular exercise. The effect of machines that produce alternate suction and pressure in a transparent boot surrounding the extremity, is variable. In acute arterial occlusion, in frostbite, and in arteriosclerosis of the large and medium sized vessels, this treatment has been of benefit. In Buerger's disease and in arteriolar obstructions seen in arteriosclerotics and diabetics, the results have been doubtful. The difficulty in evaluating the merits of this treatment lies in the fact that such patients are usually also treated by rest in bed, mild heat, forced intake of fluids, abstinence from tobacco, and vasodilators. In addition, the hospitalization of these patients enables the physician to regulate the patient's diet, and to provide proper surgical care of the wounds, ulcerations, or necroses. Finally there is a psychic influence, which should not be disregarded. These patients are in fear of losing parts of their extremity; they have tried other measures, which have failed; their hope lies in this mechanical device, which will restore the circulation to normal. One must also take into consideration that many of the elderly arteriosclerotics have cerebral vascular disturbances; they are easily impressed, are delighted or become despondent at the slightest change in their physical condition. Nevertheless from an experience with a considerable number of ambulatory patients, who have received no medication, no mental suggestions, no diets, and whose only treatment consisted of one-hour treatments by alternating suction and pressure three times a week, Mrs. Eunice Roth, Miss Elizabeth Strunk and I gained the impression that, roughly, one-half of the patients have had subjective relief.*[1] These treatments cannot influence the action of the heart nor can they change the site or extent of the vascular occlusion. They do not make new feet for the old.

Intermittent venous hyperemia. In analyzing the mechanism by which alternating suction and pressure exert their effects on peripheral circulation, Hick, Coulter and I[2] were struck by the fact, that with each period of suction, there occurs constriction of the thigh at the cuff, which is released during the positive phase. The phenomena of reddening of the toes, filling of the veins, rise of surface temperature, and the increased mobility of the toes, all of which can be observed as objective changes produced by passive vascular exercise, can be duplicated by intermittent venous hyperemia. While any blood pressure apparatus can be used to produce intermittent venous stasis, a wide, eight-inch cuff, conically shaped to fit the thigh is preferable. The amount of pressure should not exceed the diastolic pressure of the extremity at that level. It usually varies between 90 and 60 millimeters of mercury. If the extremity is edematous, cyanotic, ulcerated or gangrenous, 40 millimeters of mercury should not be exceeded, although with later improvement the pressure can be gradually raised. The duration of venous compression is determined by the appearance of definite redness; this occurs in from one to two minutes, when pressures of 60 to 90 millimeters are used. The duration of the release should be as long as that of the compression.

*In a recent survey, Beck, Roth and I found that the degree of involvement present at the onset of treatment, determines the benefit one may expect from such therapy.

The effect of such a procedure is a mechanical filling and stretching of the vascular tree during constriction and a chemical vasodilatation during release as a result of an oxygen-debt, which develops during constriction. Elevation on a pillow helps to empty the dilated vascular tree by gravity and thus supplants the positive phase in the suction and pressure treatment.

Such treatment can be used at home, at comparatively little cost, far away from medical centers. It is therefore available to a large number of patients. I have not been able to find any difference between the clinical results obtained by this method and the suction-pressure treatment.

Recently an apparatus has been described with an automatic control of constriction and release. Patients may be treated at home, and in suitable cases, may continue their occupation. Some patients have been faithfully using this treatment at home as long as two and three years.

Medication in Peripheral Vascular Disease

Provided that cardiac function is adequate and blood pressure is maintained, the object of drug therapy in peripheral vascular disease is to dilate that peripheral vascular bed. This can be attained by measures producing central vasodilatation through fever, through mechanical stretching, by increasing blood-volume, or by drugs acting on the smooth muscle of the blood vessels.

Typhoid vaccine. Injections of typhoid vaccine have been made to produce fever and thereby obtain peripheral vasodilatation for many years. It is customary to start with 25 million bacteria of a triple typhoid vaccine intravenously and increase the dose up to 300 million bacteria. A course of treatment consists of about 12 injections given biweekly. It must be remembered that arteriosclerotics, especially with cardiac impairment, are endangered by such treatment, and that even in Buerger's disease, where its use is especially recommended, the chill preceding the fever is accompanied by vessel spasm and may produce thrombosis. It has also been shown that the fever thus produced is central in origin, and that vasodilatation cannot be produced by it in a sympathectomized extremity. My practice has been to avoid these large doses and to establish one which will produce only a minimal rise in temperature and white count without a clinical reaction. These subreactionary doses vary between one million and one hundred thousand bacteria, given intravenously and may be cautiously raised, so that they never produce a clinical reaction. It is thought that the action of such treatment is not so much the production of fever as its stimulating effect on the low-grade, chronic inflammation of the arterial wall; perhaps it may also act as a non-specific desensitization of a sensitized intima. At all events, the clinical results have been just as good in patients suffering from Buerger's disease, as when using the larger doses, and the treatment is convenient and harmless. Only inflammatory lesions have been treated by this method.

Hypertonic solutions. Many authors place great emphasis on intravenous injections of a 5 per cent solution of sodium chloride, given in doses of 150 to 300 cubic centimeters, two or three times a week. One can actually demonstrate an increased pulse volume of an extremity during and at the completion of the injection. This treatment is mainly advocated in Buerger's disease. Three hundred cubic centimeters of 5 per cent salt solution should attract 1200 cubic centimeters of water from the tissues, in order to reestablish the isotonicity of the blood. This increase in blood volume in patients, whose blood volume is low and whose blood viscosity is usually slightly increased is beneficial. In my experience, however, the mechanical effect of this increased blood volume disappears in one hour. A possible slight hemolysis, which one might get from such hypertonic solutions, could act as a mild parenteral protein reaction, but this can certainly be accomplished in a simpler manner by small doses of typhoid vaccine. Furthermore, one might consider the dehydrating effect of hypertonic solutions on edematous tissues and on moist gangrene, but should such an effect be desirable, 50 per cent dextrose or sucrose would be much more efficacious. In spite of the emphatic insistence of many authors, I have not found it necessary to institute these intravenous injections routinely. The effect on blood volume is so transitory, and their other effects are so much more simply obtainable, that the injections have been abandoned in my clinic. That water is forced in all patients to 12 glasses a day, unless contraindicated, is obvious.

There is a possible effect of the sodium or the sodium chloride on Buerger's disease. Should one feel the necessity of supplying it to the

patient, one can readily give ten fifteen grain tablets of sodium chloride or Ringer's tablets by mouth. This at least will make the patient more thirsty and will also retain fluid longer in the body, than if water alone is ingested. Diets, low in potassium, accomplish the same effect.

Alcohol. The sedative and vasodilator action of moderate doses of alcohol is well known. Actually it is easy to demonstrate the rise of the temperature of a toe and the increased oscillometric readings following intake of an alcoholic beverage. For those who are used to taking a highball or a cocktail before dinner temporary increase in circulation can be agreeably obtained. Whether it has a slowly accumulating beneficial effect on the reserve capacity of the vascular bed is doubtful. At least one can hold out one compensation for the complete abstinence from tobacco, on which I strenuously insist. In fact, patients are not accepted for any form, physical, medical or surgical treatment, unless they are willing to stop smoking entirely. This rule applies to Buerger's disease and is less rigidly enforced in arteriosclerotics.

Theobromine. This group of alkaloids has been used by our group since the emphasis of George Scupham on its beneficial effects. The alkaloid, or the sodium salicylate or sodium acetate is prescribed three times a day in doses of 7½ to 10 grains. It must be taken after meals or at least with some bicarbonate of soda as occasionally gastric disturbances are experienced. It can be taken for many months in succession without harmful effects and benefits impaired coronary circulation which frequently coexists.

Papaverine. This drug relaxes spasm of smooth muscle and in addition has a slight narcotic effect. While it can be used orally for a prolonged time, combined with theobromine, its greatest usefulness is in acute arterial occlusions any large vessel. I have employed doses of ½ grain intravenously in early cases of embolism and thrombosis, and feel certain of its value in maintaining and facilitating collateral circulation.

Nitrites. Sodium nitrite, in doses of one cubic centimeter of a freshly prepared four per cent solution has been recently used in our clinic to test the capacity of the vascular bed.[3] There are no untoward effects from this small dose but the patient should be kept in the horizontal position. The effect lasts at least one hour. A therapeutic application of the drug in acute arterial occlusion is unwise, as it may increase the tendency to shock.

Choline derivatives. The vasodilating action of acetyl choline and beta-mecholine are sufficiently known. They can be given intra-muscularly and even by mouth, if large doses are used. They also can be introduced under the skin by iontophoresis as discussed above. The action of this drug, which is identical with parasympathetic stimulation is, however, quite evanescent. The same is true of prostigmine.

Iodides. Iodides have been used empirically in arteriosclerosis for a long time. Its effect on gummatous lesions is well known. Evidence that it reduces blood viscosity or that it links itself with blood lipoids and, therefore, inhibits lipoid deposits in the early stages of human atheromatosis is not conclusive. It has been shown that both iodine and thyroid extract prevent experimental atherosclerosis produced by feeding of cholesterol to rabbits. Clinically the intermittent administration of iodides to arteriosclerotics and also to patients with Buerger's disease may be of benefit.

Surgical Treatment of Peripheral Vascular Disease

Indications for sympathectomy in peripheral circulatory disturbances have been summarized in the accompanying table (table 4). For a

TABLE 4 — **Indications of Sympathectomy for Peripheral Circulatory Disturbances**

Diagnosis	Indications
Raynaud's Phenomena	Lack of marked structural changes in the vessels; absence of sclerodactylia
Buerger's Disease	Definite collateral reserve. Absence of acute inflammation or arteriolar destruction. Poor response to conservative treatment.
Poliomyelitis	Moderate paralysis limited to one extremity; evidence of vasospastic phenomena. Age preferably between six and ten.
Reflex Dystrophy (Causalgia, traumatic vessel spasm, Sudeck's atrophy)	Severe cases, resistant to physiotherapy exhibiting exaggerated vasomotor responses

sympathetic denervation of the upper extremity, the preganglionic sympathectomy of Smithwick[4] has superceded all previous methods. For the lower extremity, the lumbar ganglionated trunk between the second and fourth ganglia is excised. An anterolateral extraperitoneal approach is used. The causes of failure are readily found in incomplete denervation, improper selection of the type of patient or postganglionic degeneration. Our later operations are undoubtedly more successful than the earlier ones.

Obviously other forms of management should be combined with surgical treatment to obtain the best results. The patient with Buerger's disease, who reverts to smoking will suffer thromboses in the sympathectomised extremity. The outstanding effect of sympathectomy on peripheral circulation is that it frees the extremity from vasomotor control, and prevents all intrinsic and extrinsic stimuli from producing vasoconstriction. At the same time the metabolic control of the vascular supply, the direct action of heat and cold, and the reactive hyperemia produced by anoxemia remain intact. Numerous observations have convinced me that after sympathectomy the circulation does not return to the preoperative state.

Paravertebral block with procaine for the relief of vessel spasm which occurs following acute vascular occlusions. A temporary block of the sympathetics with procaine is a most efficient procedure. It requires special equipment and a certain amount of experience. Together with papaverine and large abdominal heat cradles it constitutes a most efficient method to prevent gangrene in cases of sudden thrombosis and embolism.

Paravertebral block with alcohol. The thoracic and lumbar sympathetic trunks and ganglia can be injected with 95 per cent alcohol, which duplicates the effect of sympathectomy for at least 4 to 6 months. A rather extensive trial of this method in our clinic indicates that: (1) the injections even in skilled hands result in a certain percentage of neuralgias, in the thoracic region, (2) because of the anatomic variations, or lymphatic enlargements the injections do not always produce a complete sympathetic denervation, but (3) in patients who are too debilitated or whose condition does not warrant sympathectomy, the method is an excellent substitute. In patients with cold, numb, pulseless extremities, following arterial embolism, in younger arteriosclerotics, and older patients with Buerger's disease, the treatment relieves rest pain, tingling, and numbness. It will not improve intermittent claudication, but will place the extremity in a state free from environmental influences.

Peripheral nerve block. In the presence of continuous intractable pain of the toes or foot, with or without gangrene, desensitization of the foot can be obtained by crushing the five sensory nerves of the foot with a hemostat. No motor paralysis results when these operations are done a few inches above the ankle, except that of the small muscles of the foot. This causes relaxation of the transverse arch, which must be supported by padding. In bedridden patients, who refuse amputation or whose condition does not warrant it, it affords great relief, facilitating avoidance of narcotics. Ulcers and gangrenous areas can be dressed and dakinized without pain. In the ambulatory patient, whose foot is entirely insensitive, there is some danger of injury remaining unrecognized. Calluses may become infected and infection may spread under the plantar fascia before the patient reports to the surgeon. While reactive hyperemia is present in a completely denervated extremity, so-called "trophic" ulcers may develop because the short axon reflexes from sensory nerves to blood vessels are lost. Nevertheless, I have removed toes and obtained primary union in such anesthetized areas.

One important point must be made. Should nerve crushing be decided on, it is advisable to desensitize the entire foot in two or three stages. Desensitization of a part of the foot, almost invariably leads to complaints in the unanesthetized areas. There is also an occasional patient who will continue to have deep, boring pain — though the skin is entirely numb above the same area. Whether this is "central" pain emanating from higher centers that have been subjected to continuous painful stimuli, or whether these sensations are carried along the blood vessels for longer segments, is insufficiently understood. Unfortunately the surgeon often gets these patients at a time when they are addicted to narcotics, which constitutes an additional problem.

Amputations. When parts of the extremity are irrevocably lost or endanger life by absorption and spreading infection, delay of amputation is dangerous. With the advent of newer methods of conservative therapy, the impression has spread among physicians and the laity that amputations are unnecessary mutilations and that the up-to-date doctor does not amputate. Aside from the fact that some patients require immediate amputation because of spreading infection or massive gangrene, one often sees cold, pulseless, paralyzed legs, which have been "saved," but which are useless to the patient and cause continuous pain. Such patients can be restored to economic efficiency by amputation at an optimal level. This level must be determined by the level of adequate circulation and by considerations of adequate weight bearing and other technical requirements of a well fitting artificial limb. The technic of amputations has been so modified that these are now delicate plastic operations performed under low spinal anesthesia and should minimize the appearance of painful cold stumps with stump neuromas, ulcers, marginal necrosis of bone and a paralyzed musculature.

CONCLUSIONS

This brief survey describes the experience of our clinic in the treatment of peripheral vascular disease and does not aim to cover the entire field. Perhaps the most important step to obtain best results — after the proper diagnosis is made — is to strike a proper balance between these various forms of therapy. Anyone who extolls the virtues of one form of therapy to the neglect of others, will ultimately have to admit that a balanced therapy leads to the best results.

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CONSERVATIVE USE OF ARTIFICIAL PNEUMOTHORAX

GEORGE H. VERNON, M.D.
SPRINGFIELD

Artificial pneumothorax along with other forms of collapse therapy in the treatment of pulmonary tuberculosis has achieved widespread acceptance. This recognition of its value has led to abuses with which we are all familiar. Routine use in minimal cases and the encouragement of ambulatory treatment with patients continuing at work, are practices which are mentioned only to be condemned.

The stress which has been placed on the mechanical forms of rest has caused many to forget the importance of simple and complete bed rest. Frequently one must defend his position for allowing the spontaneous recovery of a tuberculous patient. This is particularly true if the process involves six months to a year of complete physical inactivity. Sight is lost, of course, of the fundamentally important fact, that such a program should be followed even in those patients who require collapse measures. This attitude has been more prevalent among the younger generation of tuberculosis physicians. A note of conservatism coming from one of this generation may therefore be taken as a hopeful sign. The background for these remarks is the experience of over seven years in active charge of the pneumothorax work in a small private sanatorium. During this time it has been my good fortune to have the conservative supervision of Dr. George Palmer.

The figures to be presented are not considered to be statistically important. They deal with the five year period from January 1, 1935 to January 1, 1940. In a group of 74 patients, the treatment had been started elsewhere in 29, and 6 were seen as outpatients only. This leaves 46 in whom the decision and attempt to establish artificial pneumothorax was made by us. It is with this group, which amounts to roughly 25 per cent of all patients treated, that we are at present concerned. This proportion was influenced by two tendencies, first to allow a chance for natural resolution and second a hesitancy to interfere with the apparently inevitable outcome in advanced bilateral disease.

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The estimated duration of active tuberculosis as determined by the history was less than six months in 18 instances and from six months to one year in an additional 12, so that in about two thirds of the total it was less than a year. The period of observation in the sanatorium prior to the institution of pneumothorax therapy was much shorter. In over 50 per cent of the cases it was less than one month. This was an interesting and somewhat surprising finding. We have always felt that artificial pneumothorax was decidedly an elective procedure, and one not to be undertaken lightly. We agree with the recently expressed view that, "pneumothorax as a treatment, in its long-time implications, is as serious as thoracoplasty."[1] The size of this group was increased by the number of patients admitted primarily for such therapy after the failure of spontaneous improvement on rest elsewhere. Others had had repeated hemoptysis or active pulmonary hemorrhage and collapse of the lung was a more or less emergency procedure.

Consideration of the extent of lung involvement reveals an overwhelming majority with far advanced disease. Only 4 minimal cases were included and in 3 of these it was by the replacement of a pleural effusion where the evidence of parenchymal involvement proved to be slight. Seven with moderately advanced and 35 with far advanced lesions were seen. Of the 35 far advanced cases, 24, or about half the total number treated had severe constitutional symptoms at the time collapse therapy was undertaken.

In classifying the most urgent indication, 10 had predominantly unilateral disease without definite evidence of cavitation. Included here were those whose lesions were stationary or progressive on rest alone. Twenty had obvious excavation which in our judgment was unlikely to heal by natural resolution or which had failed to show improvement under observation. In 12 instances recent hemorrhage had occurred. Four pneumothoraces were instituted by the aspiration of pleural effusions and their replacement by air. These were followed carefully to determine whether the collapse so established should be continued as a therapeutic measure.

Consideration of these figures reveals several important principles. Most minimal and many moderately and far advanced cases make rapid and progressive improvement on properly supervised bed rest.[2] When one allows this opportunity it naturally follows that the number of minimal and moderately advanced lesions in the collapse group will be small. The danger of spread of the disease during a reasonable period of watchful expectancy has been very much exaggerated. We feel that where it occurs frequently the rest program has been weakly conceived and poorly executed. Less can be expected from the patient with open cavity and marked constitutional symptoms. Such individuals should have active treatment earlier and should constitute the bulk of a well regulated sanatorium pneumothorax service.

In this study it was found that the right lung had been chiefly involved in 19 cases and the left in 27. Of 47 attempted inductions, 7 were complete failures in that free intrapleural space could not be found. Factors which militate against success include widespread involvement, history of pleurisy, disease of long standing and proliferative in type and patient in the upper age groups. In an additional 12 the anatomical result was poor, usually due to extensive adhesions. Five of these might well be added to the group of complete failures since they were abandoned within less than six months, being felt to be either ineffectual or actually harmful. In 6 the collapse was fair and in 22 it was good from an anatomical point of view. In brief, the results were considered good in slightly less than one half, and adequate to justify the continuation of the treatment in about two thirds of the total.

During the period in which the patients were under observation the tension within the intrapleural space was classified as negative, intermediate or positive. It was found that only negative, or less than atmospheric pressures had been used in 8 instances. In 13 they were intermediate and in 18 greater than atmospheric. Many factors enter into the determination of the intrapleural pressures used in a given case.[3,4] The ideal situation is that in which satisfactory results are obtained while remaining entirely on the negative side. Here the process is one of relaxation and will usually be selective if there are no adhesions and the disease is localized.

Excessive pressures used early in the presence of widespread adhesions and exudative disease are very dangerous. Such attempts to force the

establishment of collapse by compression, and against adhesions, are misguided and can only lead to serious complications and poor results. There is a point, however, in the evolution of many pneumothoraces, where adequate collapse has originally been obtained with negative or intermediate readings when a tendency to re-expansion of the lung becomes evident. In such instances positive pressures can and should be used to slow down or stop the premature loss of the pneumothorax space.

The above discussion is based largely on anatomical considerations, and it is necessary constantly to remind oneself that tuberculosis is an infectious disease. Mere change in the position of tuberculous lesions does not automatically and immediately effect their transformation into healed scars. Even the coaptation of cavity walls, as desirable as this may be, does not in itself cure the patient. What is required is the acquisition by the body of the little understood factors of increased resistance and immunity which enable it to stop the spread of the infection. Allergic and immunological adjustments must be made so that the reactions become predominantly those of proliferation and fibrous tissue formation in contrast to the previous exudative, ulcerative, caseative tendencies. These changes require time. It is undoubtedly true that much of the value of early relaxation of the diseased lung by artificial pneumothorax is based on the following facts. The lung is put more completely at rest. Absorption from the tuberculous lesions is decreased. Their spread, especially to the better side, is usually prevented. These conditions are operative for a sufficient length of time to carry the patient over a critical period and allow his natural bodily reactions to change and develop to the place where progressive healing is possible.

One measure of the success of artificial pneumothorax is the conversion of the sputum. Bacilli have usually been present intermittently or continuously in the cases so treated. Sputum conversion was considered to have occurred at the time of the first negative which was maintained for three months, and which was seldom or never positive later. In 22 patients, or a good 50 per cent of those in which any collapse was obtained, the organisms disappeared within the first three months. In no instance did this occur after the end of a years treatment. This is in agreement with common experience in that it shows that the good results are usually fairly prompt in making their appearance.[5] It is another argument against the prolonged maintenance of unsatisfactory pneumothoraces, a practice which is frequently harmful in itself and which delays the possible benefits of other measures.

The occurrence of fluid in the pleural space is so common that it can hardly be considered as a complication. Success or failure is frequently determined by the management of the situations which arise in this connection. It is true that the formation of a clear straw colored effusion will usually slow up the absorption of air and cut down on the amount and frequency of refills required to maintain the desired collapse. However, the rather severe constitutional reaction, which may accompany its formation, cannot be minimized. When one realizes that every such patient is a potential candidate for a tuberculous empyema, he must feel more comfortable about those whose pleural spaces remain clear.

Published series show that between 50 and 80 per cent of cases develop demonstrable effusions at some time during collapse therapy.[6] Of these 20 to 30 per cent go on to the formation of tuberculous empyema. The figures which follow should be discounted because in most instances they cover only part of the course of treatment. While it is true that effusions are more likely to develop during the early months, they may occur at any time and particularly during attempts at reexpansion after prolonged collapse. Twenty-two patients remained free of fluid during the period of observation. Seventeen had an appreciable amount and practically all were aspirated on one or more occasions. In 6 of these the effusion was present at the beginning of treatment and the pneumothorax was started by aspiration and replacement with air.

Fluid is viewed rather complacently by some workers, and only disturbed when present in large amounts or accompanied by marked constitutional symptoms. Our policy has been frequently repeated aspirations where fluoroscopy indicates an estimated 100 c.c. or more. This permits changes in the manometer readings through which by trial one may determine what intrapleural pressures will hinder its recur-

rence.[7] The regular removal of this potential culture medium cuts down the chance of empyema formation. It is also true that permitting its accumulation over periods of time leads to marked pleural thickening and the formation of much fibrinous debris. In the cases treated with frequent aspirations during the five year period in question only one developed a typical tuberculous empyema.

A factor of great importance in the final outcome of pneumothorax treatment is the extent and evolution of the disease in the contralateral lung. Among 35 patients, the involvement of the opposite lung was minimal or undetectable in 24, moderate in 10, and advanced in 1. In all of these there was satisfactory regression of the lesions. The remaining 4 require special mention. The first had bilateral pneumothorax. The second had temporary collapse for control of hemorrhage, and died of tuberculosis and Parkinson's disease. Another refused collapse of a large cavity, suffered massive hemorrhage which was treated by artificial pneumothorax, and died within three months with widespread new lesions in the better lung. The fourth, a male 54 years of age, failed to respond to an anatomically good collapse, left the sanatorium to have refills continued at home and died within one year.

Bloch, Tucker and Adams have recently pointed out the need for longer periods of observation in reporting results of pneumothorax therapy.[1] They suggest a minimum of two years following complete reexpansion, before feeling reasonably sure of a favorable outcome. Their position is well taken and emphasizes one of the difficulties in evaluating therapeutic methods in tuberculosis. In view of the relatively short time covered by this study, little can be indicated as to what will finally be accomplished in many of these patients. However, their condition, where known by personal observation, has been classified at yearly intervals following the institution of collapse therapy. This is not necessarily their present condition as in some instances they have been lost to view. Two have been followed for less than a year. The condition of 6 was unknown at the end of one year. Two were dead, 1 improved folowing thoracoplasty, and 1 with reexpanded lung and arrested disease. Nine were still under treatment, 1 with tuberculous empyema, 2 improved, 5 quiescent and 1 arrested. Among 11 patients after two years, 1 was dead; 2 had reexpanded lungs, 1 being improved and the other arrested; and 8 were receiving refills, 1 improved, 3 quiescent and 4 arrested. Two patients with arrested lesions were still taking air at the end of three years. In the four year group, 1 was dead, 1 had bilateral pneumothorax, and 2 were reexpanded and arrested. Five years later 1 was reexpanded with arrested disease.

Among those in whom unsuccessful attempts to establish pneumothorax had been made; 1 died within a year, 2 had subsequent phrenic operations and died within two years, 2 had subsequent phrenic operations and thoracoplasties and are still receiving sanatorium care, and the whereabouts of 2 were unknown at the end of a year.

It is interesting to note that in no instance has elective reexpansion been undertaken where satisfactory collapse of an originally advanced lesion could be maintained.[8] The pneumothorax space was lost unavoidably in 5 cases, usually due to obliterative pleuritis. There was elective reexpansion in 5, due either to unsatisfactory collapse or upon advice of other physicians. Of the 29 remaining, 11 are still under collapse personally, 6 are receiving refills elsewhere, 4 are dead and the whereabouts of 8 are unknown.

SUMMARY

Results of five years experience with artificial pneumothorax in a group of 46 tuberculous patients have been presented.

George H. Vernon, M.D.,
Springfield, Illinois.

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POTENTIAL INGUINAL HERNIA WITH ESPECIAL REFERENCE TO DIRECT HERNIA

HILLIER L. BAKER, M.D.
Rush Medical College, Presbyterian Hospital
CHICAGO

A questionnaire addressed to the heads of medical departments of several large corporations on the question of inguinal hernia as a cause of disability, brought the observation that all males were considered as having potential hernias. This attitude was adopted because the surgeons questioned felt that an indirect hernia was primarily the result of a congenital preformed sac, while the direct hernia was due to either developmental defects in the muscular structures of the inguinal region, or to atrophic changes in the musculature due to age, obesity or other factors.

In examining applicants for inguinal hernia, the criteria for refusal to employ varied. The presence of enlarged external and internal rings, the finding of a large external inguinal ring with an impulse on coughing, and the presence of an enlarged external ring alone. Their criteria as well as the age and type of work in which the employee would be engaged, were factors in passing or rejecting the applicant. The most liberal interpretation was that taken by an examining surgeon who said that he did not reject any applicant for employment who did not have a visible or palpable "bulge" when the abdominal pressure was raised by straining or coughing. He stated that the incidence of hernia was not any higher amongst the many thousands of his company employees than those in a similar line of work, when the criteria for examination was more strict.

In a group of patients with unilateral inguinal hernia either of the direct or indirect type in which the cause of the hernia was in many instances ascribed to occupational trauma; the unaffected side was operated upon.

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This was done because the patient had an enlarged external inguinal ring, or large internal ring on the unaffected side. The patients were not thought to have bilateral hernia. The unaffected side was operated upon because it was felt that the patient had anatomical defects similar to that which had caused the hernia on the affected side and that a potential hernia was present, which if corrected would prevent future disability and hospitalization. None of these patients were in public or charity hospitals. Consequently it has been possible to check on the results of the operative procedure. The item of interest in this study has been the high incidence of direct hernia, and the fact that anatomically the factors making for the production of direct hernia is present at an early age and is, as is well known, a bilateral defect.

There were thirty-one males in the series. The youngest was four years of age and the oldest sixty-four years of age. Eighteen had hernia on the left side while thirteen had right sided hernia. There were fourteen indirect hernias. Eight had indirect and direct hernias. Two had sliding hernias. Five had direct hernias and two had indirect hernias with non-descent of the testicle.

The conjoined tendon was fragmentary or poorly developed in twenty-one patients. The poor development of the conjoined tendon was bilateral in sixteen patients and this occurred in patients twenty-nine years of age or older. In three patients the conjoined tendon was poorly developed on the side in which the hernia was present but well developed on the unaffected side. In these patients the hernia which was found was large and had been present for many years.

Operation on the unaffected side showed eight non-obliterations of the tunica funicularis, three small indirect and direct hernias, eight small direct hernias, one hydrocele of the cord. In eleven patients no evidence of hernia was found. Seven had well developed conjoined tendons and five poorly developed conjoined tendons. The conjoined tendon was poorly developed or fragmentary in nineteen cases. In a boy of five years, on the unaffected side the conjoined tendon was poorly developed and a small direct hernia was present, while on the opposite side he had a large indirect hernia with a well developed con-

joined tendon. Two men, twenty years and twenty-seven years respectively, had poorly developed conjoined tendons on the unaffected side, but well developed on the hernial side.

In this series of patients the most constant finding was a poor development of the conjoined tendon. This defect was most constant in men over thirty years of age, in those that had direct inguinal hernia. The defect in the conjoined tendon was found unilaterally in patients who had large indirect, or sliding hernia of long duration, and was also found in a boy six years of age.

Direct hernia has for predisposing factors, defects in development of the transversalis fascia and conjoined tendon. Atrophy of muscle due to age, obesity or disease. The exciting cause occasionally is the strain of heavy lifting by one of sedentary occupation; in this instance, the transversalis fascia suddenly gives way.

In the majority of instances direct hernias are slow of development, hence we find them in men who have done heavy labor for many years. Patients with chronic bronchitis or prostatic enlargement with coughing and straining to which they are subject also, often develop direct hernias.

Indirect or oblique hernia as has already been mentioned, is due to the presence of a congenitally performed sac. This type of hernia as we know may be present at birth. The sac may become closed, to reopen in later life, because of sudden or violent straining.

The repair of an indirect hernia anatomically is quite different than that of a direct hernia. But as an indirect and direct hernia are frequently a part of the pathology present, the repair of either or both must be contemplated at the time of operation.

A brief review of the anatomy of the inguinal region shows that the funicularis spermiatious as it passes from the abdominal to the external cutaneous ring is covered by the aponeurosis of the external oblique muscle. At the external cutaneous ring the arching fibres of the external oblique aponeurosis are inserted into the pubic tubercle, the pubic crest and symphysis of the pubis.

In well developed muscular individuals the cord structures lie in a cleft of the muscle fibres of the internal oblique. Beneath the internal oblique are the transversalis muscle and fascia.

The inguinal triangle through which a direct hernia occurs is normally protected in muscular individuals by a stout transversalis fascia, the arching fibres of the internal oblique and transversalis muscles and is further re-enforced by a fusion of the fascias of the internal oblique and transversalis muscles. This fused fascia, the falx inguinalis or conjoined tendon is inserted into the front of the pubis, the pubic tubercle, and the ileopectineal line. This fused facia passes medially and with the aponeurosis of the external oblique becomes the anterior sheath of the rectus abdominus muscle.

In individuals with poorly developed musculature the falx inguinale or conjoined tendon is deficient both in its muscular and tendonous portions. The tendonous attachment to the pubis is often fragmentary or entirely missing. The transversalis fascia instead of being a firm dense fascia, is thin and meshed. These individuals have all the anatomical defects which are favorable for the development of a direct inguinal hernia. The developmental defects mentioned are studied best on patients undergoing herniotomy, and they are seen much more clearly on the living than on the cadaver. This is particularly true in respect to direct hernia.

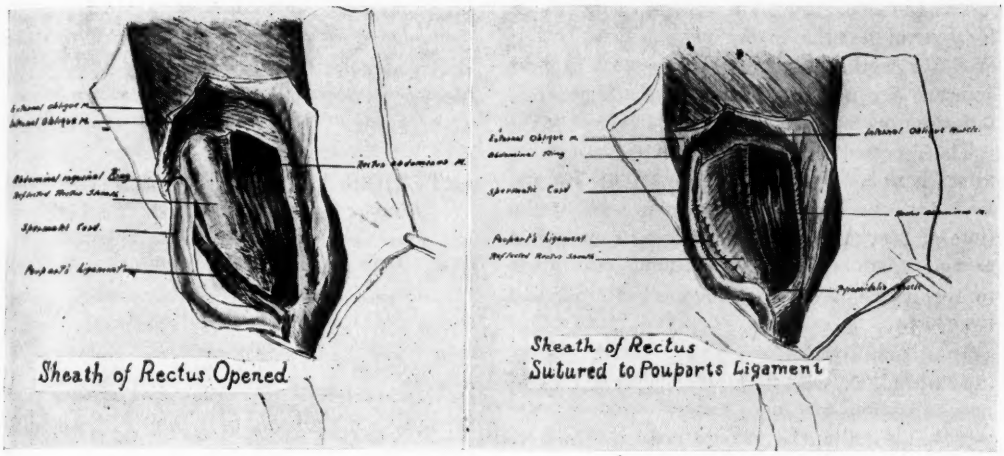
Types of Direct Hernia:

1. The simplest type of direct hernia is caused by a small hole in the transversalis fascia through which may protrude properitoneal fat or a portion of the wall of the urinary bladder. This type is comparable to a hernia of the linealba.

2. The direct hernia sac may be an enlargement of an indirect inguinal hernia, or it may be a part of a sliding hernia of the sigmoid or cecum.

3. The globular or ovoid type of direct hernia is the most usual type. The transversalis fascia has been slowly stretched or torn. The swelling may extend as far as the external ring in large hernias.

In the repair of direct inguinal hernia an adequate skin incision is made to insure good exposure. The external oblique is incised and the upper and lower edges retracted. The cord is identified and examined for evidence of an indirect sac. The direct hernial sac is easily separated from the cord and the cord retracted. The defect in the transversalis fascia may be closed



if the hernial opening is small. In a majority of cases, however, the fascial defect is too large to be closed without tension on the suture line.

Opening of the direct sac is of doubtful value. Injury to the inferior epigastric vessels or opening the urinary bladder may follow unless the sac is opened in the region of the internal ring. When a direct hernia co-exists with an indirect hernia, the sac then is opened. The preceding precautions being kept in mind.

Repair of the fascial defect is best accomplished by fascial approximation. Suture of a poorly developed conjoined tendon to the inguinal ligament is unsatisfactory because muscle is approximated to fascia and as has been pointed out by Seelig and Chouke, the suture line is likely to be unsatisfactory. Then too, tension on the suture line often defeats the purpose of the closure and results in recurrence.

A fascial closure of a direct hernia can be done by splitting the sheath of the rectus muscle. The closure is physiologically and anatomically correct because fascia is approximated to fascia without tension.

The rectus sheath is opened from the pubic crest in a curved direction upward and outward toward the region of the internal ring. With the cord retracted an anchoring suture is placed through the periosteum of the pubic spine and the rectus sheath is anchored to it. Then by a series of interrupted sutures, using silk or catgut the reflected fascia of the rectus sheath is approximated to the inguinal ligament. The external oblique may be approximated beneath the cord as a reinforcing measure. Or the upper leaf may be approximated to the inguinal ligament and the lower leaf may be used to cover the cord. Care should be taken that the newly made internal ring be not too tight so as to constrict the cord or ilioinguinal nerve. In this manner a firm fascial wall is constructed without tension and the defect in the inguinal triangle is closed.

The after care of patients operated upon for direct hernia is as important as the operative procedure itself.

The use of local or spinal anesthesia is desirable. Because, 1. During the operative procedure by having the patient strain or cough, the defects of the transversalis fascia can be seen more easily. Then too, the irritation to the lungs by inhalation anesthesia can be avoided. This is important, because coughing or straining may cause a break in the recent repair.

2. The patient should be put to bed in the "jack-knife" position. That is with the back and head elevated, and the knees flexed and supported by pillows. This position relieves the strain on the suture line. Deep breathing exercises and movement of the extremities should be instituted early. This may reduce the incidence of pulmonary embolus.

3. Reflex urinary incontinence and abdominal distention are occasionally troublesome. The use of prostigmine, and heat to the abdomen, often obviates catheterization.

4. The patient should be kept in bed for at least twelve days. A support, such as the "London Type," should be worn when the patient walks.

The continued use of a support is advisable for several months if the patient does lifting or heavy work. Return to heavy work is best deferred for a six months period if possible, following operation.

The reported high incidence of recurrence in direct hernia, is I think, due to several factors. In many, the direct hernia was present at the time of previous operation, but was over-looked or not recognized, hence, no attempt was made to repair the defect, or the method used was incorrect.

In others, a repair was made with tension on the suture line. The necessary length of time of hospitalization was not insisted upon, or was refused. Finally the patient was returned to work at heavy labor too soon after operation.

SUMMARY AND CONCLUSIONS

1. Patients with unilateral hernia frequently have similar defects on the unaffected side. The conjoined tendon, is fragmentary in the majority of cases and the defect is bilateral.
2. Non-obliteration of the process funicularis is not an inconstant finding.
3. The use of local anesthesia reveals hernial defects when the patient increases intra-abdominal pressure. These defects might otherwise be overlooked. This is true especially for small direct or indirect hernia.
4. Direct hernia is a common bilateral defect and the repair can be best affected by utilizing the sheath of the rectus muscle.
5. Patients with large external or internal inguinal rings, with unilateral hernia, should have the unaffected side repaired. This may prevent future disability and hospitalization.
6. After care of patients following a herniotomy, is important. Bed rest for at least twelve to fourteen days should be insisted upon. A hernial support should be worn and the patient should be put at light work and if heavy labor must be done, the patient should not return to this type of work for at least four months. The reported high incidence of recurrence in direct hernia is due to many factors.
   a. Non-recognition of the defect at time of original operation.
   b. Improper method of repair.
   c. Non-observance of proper post-operative care.

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ARTERIO-SCLEROTIC HEART DISEASE

ROBERT S. BERGHOFF, M.D., F.A.C.P.
Clinical Professor of Medicine Loyola University School of Medicine
and
Chairman of Scientific Service Committee Illinois State Medical Society
CHICAGO

Arterio-sclerotic or senile heart disease is an intriguing subject, not only to us of the medical profession, but to all men and women in the latter decades of life. I call your attention to the fact that as human life becomes progressively longer due to the elimination of many diseases, the control of others, the improvement of the general social and economic states of our people (due in part to a paternalistic government) heart disease assumes a constantly more formidable role, so that today it is the outstanding cause of death in adult life. To make this subject more personal, I say to you, that if we reach the seventh and eighth decades of life three out of five of us will die of one or the other of the two types of heart disease. I propose to discuss with you at this time.

Arterio-sclerotic heart disease is not strictly speaking a disease at all, but merely part and parcel of the process of growing old. It implicates and involves either the heart muscle itself or its nutrient vessels the coronaries, or quite frequently, both. Accordingly let us consider the subject under two headings — 1. Heart Muscle Disease, 2. Coronary Disease.

Heart Muscle Disease. Here we encounter at the very outset difficulty in nomenclature. The old and long-accepted term Chronic Myocarditis was found unsatisfactory and has accordingly been discarded, for an "itis" assumes an inflammatory process, which is not borne out clinically nor pathologically. "Chronic Mycardosis" is no more illuminative. Dr. Henry Christian some years ago suggested "Chronic Non-Valvular Heart Disease," appreciating the non-existence of heart muscle disease in these senile hearts.

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My associates and myself like to think and speak of these senile heart muscles as "Chronic Myocardial Insufficiency," heart muscle not diseased but simply chronically insufficient to meet the demands imposed upon it. However irrespective of the nomenclature employed, what is the clinical picture of such a heart, what are the earliest symptoms and signs?

Symptoms — The earliest subjective manifestations of an insufficient or failing senile heart muscle in order of their frequency are:

1. Dyspnoea — varying in degree from shortness of breath on exertion to intense orthopnoea.
2. Precardial pain on exertion or mental excitation of aortic and not coronary origin, and due to pressure and strain on an inelastic and chalky aorta.
3. Cough — of cardiac origin.
4. A heterogeneous strain of general symptoms in part at least of cardiac origin, such as, general fatigue and lassitude, amnesia, the melancholia of senility, etc.

Physical signs: The cardiac signs of senescence are few, yet important and sufficient. That they are relatively few is not surprising, since our friends the pathologists are able to demonstrate many senile hearts apparently entirely normal in size, shape and dimensions, with only a few and inconsequential microscopic abnormalities.

Abnormal physical signs most frequently and consistently encountered are, as follows:

1. An increase in the aortic dullness in the second interspace from an accepted average normal of 4½ cms., to 6 and 7 cms.
2. An increase or spread of the left ventricle.
3. A soft systolic (relative) murmur over the mitral valve, due to the increased width of the left ventricle.
4. A soft systolic murmur over the aorta (relative) due to the swish of blood through a chalky aorta.
5. Distant heart tones.
6. Alterations in rate, either a brady-cardia or tachycardia.
7. Alterations in rhythm, chiefly ectopic beats, particularly ventricular, extra-systoles, or auricular fibrillation.
8. A variable blood pressure change either hyper or hypotensive.

The symptoms and signs are usually sufficient to suggest senile heart muscle insufficiency, particularly when corroborated by the X-ray and electrocardiogram. The X-ray, through a two meter plate, the ortho-diagram or a teleroentgenogram will certify a widened aorta and a left ventricular spread. The electrocardiogram provides additional a certification through changes in the QRS Complex and P and T wave alterations.

Prognosis — Possibly no type of heart disease or heart abnormality provides a more promising field for mediate and immediate results. In Rheumatic Heart Disease we are dealing with heart valves already crippled, and similarly in syphilitic heart disease we are dealing with a pathology well established. Not so in the aging heart.

Treatment — The care of the aging heart resolves itself around two factors: 1. Mechanical support through rest. 2. Drugs.

Everything in this world is relative including senility and longevity. Accordingly we encounter senile heart muscles from the fifth decade of life to the tenth. Irrespective, however, of the age or youth of the individual when the symptoms and signs enumerated appear, treatment must be instigated and promptly. All activities, physical, mental and gastro-intestinal must be moderated to suit individual requirements.

Fatigue of any sort must be discountenanced. An afternoon rest period whenever feasible and practical is important. Hours of sleep at night must be lengthened and abetted when necessary with sedatives.

2. Drugs — There are two distinct schools of thought concerning the practicability and usefulness of digitalis in senile hearts. The one decries its use, the other advocates it. It is our opinion based on some experience, that digitalis in small and sustained doses, leads not only to a real amphoria but to distinct improvement and elimination of symptoms, and to prolongation of a useful existence.

Coronary Disease. Let us now consider rather hurriedly the second type or expression of senile ectasy, coronary sclerosis. We might approach this subject rather diffidently, in the nature of a catechism, answering some questions quite positively, others timidly and still others not at all.

Etiology — It's our opinion that the basic background for coronary disease in general is arteriosclerosis. We consider it a progressive situation which can and frequently does pass through four definite cycles as follows:

1. Angina (Pectoris)
2. An Intermediary type
3. Acute Occlusion with infarction
4. Status Angiosus (Rare)

When we come to the indirect or activating factors however, we leave more or less firm ground for the devious paths of theory:

How important are alcohol and tobacco? Are Mental activities more predisposing than physical? Personally we have several firmly established convictions, to-wit:

1. Excesses of any and all kinds, physical, mental, gastro-intestinal, alcohol and tobacco are distinctly relatively important.

2. Heredity is all important. We cannot discount the story of the prematurely senile individual at fifty with his definite coronary picture who tells us he resembles both his father and paternal grandfather, both of whom died in their fifth and sixth decades of heart disease. And who has not encountered the reverse, the octagenerian with a smile and a twinkle and a sprightly step, who boasts of a long lived ancestry?

Types — 1. Angina Pectoris or Angina of Effort offers as a rule no diagnostic difficulties. The classical story of either severe precordial pain or in the absence of pain, pressure or dyspnoea coming on after effort or mental excitation, short-lived, lasting but a brief moment relieved with or without treatment and leaving the individual none the worse for his episode, referred classically down the arms or into the jaws, due we believe, to temporary coronary spasm or at least to a temporary insufficiency of the coronary flow.

2. The intermediary type is not so simple, either as to diagnosis or to explanation of the direct mechanism. Here we have the story of typical coronary pain and reference, coming on either after exciting cause or more confusingly while at rest, only partially or not at all relieved by nitro-glycerine or other coronary dilators and lasting not a few moments but hours. It differs from Angina Pectoris very materially therefore and yet can hardly be classified as an occlusion in the absence of shock and all other manifestations associated with an acute infarction.

3. Acute Coronary Occlusion with Infarction. This dramatic type of coronary episode hardly calls for any diagnostic comment. If we remember its almost constant association with the usual symptoms and signs of shock, cold, clammy sweat, feeble, rapid and irregular pulse, its pallor and later resultant temperature rise, leucocytosis and electrocardiographic evidence of infarction, the picture is unmistakable.

4. Status Angiosus. Not too infrequently we encounter individuals, usually with the story of having run the gamut of coronary disease from repeated attacks of Angina of Effort, through the intermediary type, and into an occlusion with infarction until now terminally they are in constant pain whether active or at rest. They speak probably for a severe and pronounced degree of coronary sclerosis and a resultant narrowing of the coronary tree.

Prognosis: The outlook for the individual with coronary pain of the anginal type, on effort alone is certainly promising — with proper care. More grave is the outlook for the intermediary type, since it speaks for a more progressive coronary situation.

Both the mediate and immediate prognosis of acute occlusion with infarction has become less formidable in the past few decades. This is due probably to two factors, an earlier and more certain differential diagnosis and therefore more prompt, efficient and thorough management. Surely the mortality rate of at least twenty-five per cent only a few short years ago has dwindled to ten per cent or less.

When we come to theorize on the chances of the individual with a true status angiosus — optimism wanes and we must predicate or at least anticipate an uncertain outcome, and a short lived duration.

In conclusion, let me say to you, gentlemen — time forbids a consideration even in a superficial sense, of the big, broad subject of treatment of Coronary Disease. May I, however, give you this pertinent and personal admonition — Even as our earthly sun, as it approaches the horizon, yields its fiery noon-day redness to a mellow yellow, so may we as we reach the twilight of our lives, moderate our activities, slow our pace, furl our sails and sail calmly and peacefully into the harbor of senility.

IMMUNIZATION AGAINST COMMON COMMUNICABLE DISEASES

FREDERICK H. MAURER, M.D.
PEORIA

Preventive medicine has made, and is continuing to make, such rapid strides, that certain protective measures against disease, which we regard with favor today, may be entirely inadequate tomorrow. This is entirely as it should be. Progress in the development of immunologic procedures is a flexible story of advancement.

While some of our present accepted measures may be of definite and proven value, others are almost of an experimental nature, yet worthy of continued trial and further biological and clinical investigation. Only large numbers of scientific and clinical reports covering thousands of adequate controls can definitely fix the value of the immunizing agent in question.

In discussing this subject today, it is recognized at once that all of the material involved has been previously reviewed and discussed in hundreds of more accurate and excellent papers. I will attempt, therefore, only a brief and practical review of the more widely accepted and commonly used methods of active and passive immunization against the commoner communicable diseases.

The elementary facts, concerning the protective action of the human organism, in response to various antigenic stimuli, are well known to all. These antigens may have been introduced into the living organisms by either nature, in the form of disease producing micro-organisms or by man in the form of specific antigens. In either case there is set up in the tissues of the host, a protective, offensive, or defensive mechanism which gives rise to the production of many types of antibodies, such as bacteriolysins, agglutinins, opsonins, precipitins and antitoxins. If the human organism, through its struggle with the invading micro-organisms or its toxins, is able to elaborate specific antibodies sufficient to overcome the invading agents, then recovery occurs and an active immunity is acquired by virtue of the laws of nature.

With the discovery of each specific causative agent of disease came attempts on the part of man to develop antibodies in the animal or human host, by introducing into the tissues of the host either the specific agent, in attenuated or killed form, or its growth toxins. These antibodies may be developed in lesser or greater amount and the resultant active immunity be accordingly of shorter or longer duration.

Passive immunity, in contra-distinction, except that of the newborn, is acquired by the introduction into the human organisms, of animal or human serum, containing antibodies already generated in the donor by either an attack of the disease or active immunization with specific antigens. Antitoxin for the purpose of passive immunization was first used in 1894 in the treatment of Diphtheria.

Little progress in immunization was recorded before 1913 when Von Behring introduced toxin-antitoxin as an immunizing agent in producing active immunity against Diphtheria. In a small group of human subjects, he conclusively proved the value of his agent in producing immunity in about 85 percent of those treated, using three spaced doses of 1 cc. each.

Bela Schick[1] in the same year developed a diagnostic skin test to determine immunity and susceptibility to Diphtheria.

In applying the Schick Test, the skin test dose of 0.1 cc. diluted Diphtheria toxin (1/50 M.L.D. for 250 gram guinea pig) is injected intradermally (not subcutaneously) into the skin of the forearm. An urticarial like wheal is raised. If the test is positive, denoting susceptibility, an area of redness .5 cm. or more in diameter develops within 48 hours and gradually fades over a period of the following two to three weeks. Pseudo reactions, due to the protein content of the toxin, appear within 12 to 48 hours and fade rapidly.

The Schick Test is practically unnecessary under six years of age since most individuals show a positive reaction under this age. In infants under five to six months of age and in some susceptible people the test may be negative yet they may acquire diphtheria. Older children and adults may require a control test using heated toxin to eliminate pseudo reactions. Repeated Schick tests should be done to prevent lapses of immunity and under any epidemic conditions. Routinely a Schick should be done three to six months after immunization is completed, again before entering grade school and high

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school. This applies to all children whether receiving toxoid injections in infancy or not.

By 1918, Park[2] and Zingher had introduced mass immunization of school children in New York City using toxin-antitoxin. Disadvantages of this method were soon apparent. Freezing temperatures surrounding the toxin-antitoxin mixture caused a disassociation of the components liberating lethal amounts of the toxin. A number of deaths from this cause are recorded. There was also the danger of sensitization or serum reaction in the human from the horse serum content.

Sheep or goat toxin-antitoxin is now used, but only in selected cases and especially in subjects over ten years of age. In practice it is given subcutaneously in 0.5 cc., 1 cc. and 1 cc. doses at two to four week intervals. Immunity develops slowly, sometimes requiring one year to produce a negative Schick test.

In 1924, Ramon[3] introduced toxoid. This is produced by combining diphtheria toxin with various percentages of formalin. This product has proven more stable, easier of preparation and capable of producing a higher grade of immunity which is of longer duration, than the toxin-antitoxin mixture. This is still used and preferred by some clinicians. In children under ten years of age it is given in doses of 0.5 cc., 1 cc. and 1 cc. subcutaneously at two to four week intervals. For ages over ten years the dosage is reduced to 0.1 cc. then 0.2 cc., 0.5 cc. at similar or longer intervals. If reactions are not too severe an additional 0.1 cc. dose is given.

In 1926, Alum-precipitated toxoid was introduced by Glenny, Pope, Waddington and Wallace. This product was derived by treating diphtheria toxoid with aluminum potassium sulphate. The insoluble precipitate contains the antigen and the refined product has fifty percent less protein content than the Ramon toxoid. It also has the advantage of slower absorption, thus giving prolonged antibody stimulus. Early use of this product seemed to indicate that only one dose of 1.0 cc. was needed to produce 92 to 94 percent of immunity in Schick positive children. Further experience has led to the conclusion that one dose is insufficient to produce a prolonged or permanent immunity.

At the present time it is given to children under ten years of age in doses of 0.5 cc., 1 cc. and 1 cc. at two to four week intervals. The longer interval being preferred.

Since sixty-five percent of diphtheria mortality occurs below the age of five, and eighty percent of all cases occur between six months and six years of age, it is highly desirable to immunize early in this period. Eighty-five to ninety-five percent of newborns give a negative Schick Test which persists up to six or nine months of life. The age of nine months seems desirable to start Diphtheria immunization. Some physicians suggest and practice smallpox vaccination at an earlier date — even three months — because of the non-specific enhancement of the following diphtheria antigen stimulation.

In 1935, Ramon[4] and Frank and in 1937, Jones and Moss[5] introduced the use of diphtheria-tetanus toxoid mixture for simultaneous active immunization against the two diseases. The two toxins given together act more effectively in the production of their respective antitoxins than if given independently. Reactions are slight, as a rule, except in older children, adults or allergic individuals. The particular appeal of this combined immunization is the fact of the additional protection provided, without additional effort.

The basic immunity thus established against tetanus will persist for years, and it has been demonstrated that it is possible to speed up tetanus antitoxin production in the exposed patient by giving an additional dose of tetanus toxoid, and to increase the titer to a point far in excess of that provided by the usual prophylactic dose of tetanus antitoxin. This can be accomplished within seven to ten days after administration of the stimulating dose of toxoid. It is advisable to recommend, however, that if there be doubt as to the status of the tetanus immunity of the patient or if the exposure be of a massive type, that the prophylactic dose of 1,000 to 2,000 units of tetanus antitoxin be given.

Two doses of 1 cc. each of the combined toxoid are given subcutaneously at intervals of two to three months. In older or allergic individuals the first dose may be subdivided in 0.2 cc., 0.4 cc. and 0.4 cc. doses at two to three week intervals.

Occasionally a sterile abscess may form at the site of an injection as a result of the presence of

alum, an insoluble substance, in the tissues. This is of infrequent occurrence.

What is the duration of the immunity against diphtheria provided by these various methods? Reports from various groups, tested over a period of one to five years, vary as widely as the methods. Certainly, results with the use of alum precipitated toxoid in adequate doses and at proper intervals, have shown higher and more prolonged immunity.

Julius Sigurjon'sson[6] reported in October 1939, on a group of Schick tests done eight months and four years after two injections of Alum-precipitated toxoid. After eight months, 95.4 percent were Schick negative. After four years, only 66.3 percent were negative. The Ramon toxoid gives lower immunity duration. After eight months, 80.7 percent and after four years, only 26.12 percent were Schick negative.

Whatever the type of agent selected by the physician, the necessity for repeated Schick tests at routine age periods is apparent.

Convincing evidence of the value of active immunization against diphtheria is shown in the decline of the mortality rate for this disease. In 1923, the rate was 13.13 per cent, and by 1937, the rate had fallen to 1.46 percent according to the vital statistic records of the United States.

Passive immunity may be given to non-immunized or Schick positive exposures by injecting intramuscularly 1,000 to 2,000 units of Diphtheria antitoxin. This is an undesirable procedure — except in unusual circumstances where the exposed patient cannot be kept under close observation. The use of animal serum carries with it the ever present possibility of sensitization to foreign protein and the immediate, accelerated or delayed serum reaction.

SMALLPOX: — Dr. Jenner was the first to demonstrate the possibility of active immunization against this disease when he introduced vaccination against Smallpox. He transferred the contents of a cowpox pustule on the hand of a milk maid to a scarified area on the arm of a child, thus producing a vaccination and immunity against the scourge of smallpox.

Of the many types of vaccine and methods of application, that of the U. S. Public Health service is recommended. This is the multiple puncture method using calf vaccine smallpox virus and is as follows: —

The skin over the insertion of the deltoid is first cleansed and sterilized (preferably with acetone). Through a droplet of the vaccine on this area, five to ten punctures are made with the sterilized needle, care being taken not to puncture so deeply as to cause bleeding. The excess of vaccine is wiped off with sterile gauze. No dressing, shield or protective mechanism is applied. A primary "Take" begins in three or four days with a macula at the site of puncture gradually passing through the stages of vesiculation, and pustulation by the seventh to ninth day. Local systemic reactions may and usually do occur at the height of the "Take", but subside rapidly. Scabbing follows and by the end of the third week a moderately depressed scar results. A "vaccinoid reaction", showing the presence of partial immunity, is an accelerated take which passes through all stages and subsides within a few days, often subsiding before pustulation occurs. A "reaction to immunity", indicates either previous successful vaccination or immunity. Local induration and erythema occurs at the site within 24 hours, then complete subsidence. Vaccination may also be carried out by cross scratching or scarifying of the skin and rubbing in of the vaccine. To apply the vaccine intradermally the contents of a capillary vaccine tube is drawn into a hypo needle diluted up to 0.2 cc. with normal saline. One tenth of a cubic centimeter of this mixture is then injected intradermally in the same manner as in doing a Schick test. A similar sequence of vesiculation, pustulation, etc., occurs.

The age for vaccination is frankly any time from birth on, preferably before the age of three, since post vaccinal encephalitis rarely or never occurs before that age. Many clinicians recommend vaccination at three months or before giving pertussis and diphtheria immunization on account of the tendency of smallpox vaccinations to accelerate immunity of later immunizations.

Revaccination is recommended by the U. S. Public Health service at intervals of five to seven years until three successful takes are recorded. Revaccination at six and twelve years of age and in all susceptibles immediately on exposure should be routinely recommended.

Chick Embryo Smallpox Vaccine Virus as artificially grown by Goodpasture and Rivers has been used with certain claims of superiority, namely (1) that its purity is assured by growth in vitro (2) Systemic reactions are mild and (3) that no scar results when used intracutaneously.[7]

The objection that lack of scar would leave no objective evidence of a successful vaccination is countered by the fact that the presence of a scar does not always mean immunity to the disease. A vaccination scar should be regarded, however, as a badge of good citizenship and a mark of good judgment.

SCARLET FEVER: — Active immunization against Scarlet Fever has never been as popular nor enjoyed such extensive use as some other types of immunization. To be generally accepted and energetically used, a product should promise not only reasonably effective results, but also require few doses and freedom from marked reactions.

The Dick Test for susceptibility to Scarlet Fever was introduced by Drs. George F. and Gladys H. Dick. It is not 100 percent accurate, although a positive test indicates susceptibility in all but a small number of susceptible individuals. The Dick Test is done by injecting 0.1 cc. of diluted scarlet fever Dick Toxin intradermally in the same manner as the Schick Test. The positive reaction is denoted by an area of redness 0.5 cm. or larger at the site of injection. Even if faintly red it is regarded as positive and denotes susceptibility to scarlet fever. The test is more transient than the Schick Test and should be examined in 24 hours.

A positive reactor to the test may be rendered negative by five subcutaneous injections of scarlet fever toxin at weekly intervals. These doses range from 650 up to 120,000 skin test doses. Reactions today, on account of further purification of the product, are not so frequent, yet one or more of the higher dosages may cause marked systemic or local reactions. This adds nothing to its popularity with the general public. A Dick Test should follow immunization one week after the last dose and whenever exposure occurs.

In the thus apparently immunized individuals we have no assurance that the toxic effect or the invasiveness of the streptoccus Scarlitinae are in any way altered. Dr. J. A. Kolmer[8] has suggested the possibility of combining the five doses of toxin with a Streptococci Scarlitinae Vaccine, thus engendering antibacterial as well as antitoxic immunity.

The use of Scarlet Fever Toxin among nurses, doctors and inmates of child caring institutions has been recommended and used by many well informed investigators and clinicians in preventing the spread of scarlet fever to epidemic proportions. Before offering it to private patients, however, it is well to acquaint them with the possibility of reactions and the uncertainty of its beneficial results. In any case, is not to be given before the individual is from one and one-half years to two years of age.

For passive immunization against Scarlet Fever, the use of pooled convalescent serum is rapidly showing great possibilities. Given in 10 cc. doses to small children, 15 cc. for larger children and 20 cc. for adolescents and adults, Scarlet Fever is said to be prevented in 97 percent of all children and 85 percent of susceptible children. These results will naturally depend on the quality of the pooled serum and host resistance. It is entirely safe in administration.

The use of the sulfonamides as a prophylactic measure is controversial, the synergistic action, however, of both serum and sulfonamides, in treatment, has received favorable comment.

Whole adult blood and adult serum are of such fluctuating quality as to antibody content and require such large doses (80 to 100 cc.) that their use is limited.

Commercial Scarlet Fever antitoxin of animal origin, may be used in emergencies and under special circumstances. The immunity supplied is of short duration and has frequently in the past been associated with severe serum reactions. Recently several biological laboratories have produced more purified and more potent types of antitoxin (5,000 to 6,000 units per cc.) which are said to cause fewer reactions.

The Schultz-Charlton blanching test for diagnosis of Scarlet Fever is performed by injecting 0.5 cc. of scarlet fever antitoxin or convalescent serum intradermally into an area where the rash is most marked, preferably the lower abdomen. Read in 12 to 14 hours, a blanching at the site of injection is considered as positive. It is generally accepted as due to the local effect of the

antitoxin on the toxin. The limitations of this test are as follows: (1) A negative test does not rule out Scarlet Fever. (2) A papular type rash cannot be blanched. (3) It is useless where the rash is more than three days old (some workers report negative Dick & Schultz-Charlton tests even during the first two to three days).[9] Dr. Wm. Thalhimer,[10] using 1 cc. convalescent serum, states that the test is "extremely reliable in differentiating between scarlet and other rashes."

CHICKEN POX: — Chicken pox, while considered as more worrisome than serious, is not always a mild disease — particularly when associated with another disease such as Scarlet Fever or where resistance has been lowered by some other major infectious process or illness.

Active immunization with the vesicle fluid was suggested by Kling in 1913. Cooke, Hess and Unger have proven this as unreliable and offering less success in definite exposure than passive immunity. The fact that the vaccination actually produces a mild case of chicken pox which is in itself infectious, defeats its purpose. Convalescent serum prevention of chicken pox was introduced by Blackfan et al.[11] Of one hundred sixty-seven immunized, 13 percent developed chicken pox.

Convalescent Pooled Serum is given in 10 cc. doses intramuscularly. The donor or donors must be less than three weeks convalescent and the serum must be given within three days after exposure to be at all effective. The actual protective value varies from 91 percent to 35 percent depending on the quality of serum and time interval between exposures and injection.[12] No severe reactions are reported. The scarcity of such serum makes its wide use impractical.

MUMPS: — Mumps may not always be a disease of low morbidity and negative mortality if such complications as Meningo-encephalitis are taken into consideration. Tabor and Newman of New York have collected twenty-nine of these cases.

No method of active immunization is known. Convalescent Serum in doses of 8 to 20 cc. have been given intramuscularly but available statistics have failed to substantiate its beneficial effects.

MEASLES: — On account of the high mortality of this disease (2.6 percent for ages 1 to 5), the high communicability, and frequency of complications, such as pneumonia; there has been a large amount of investigation done in search of the specific agent and effective immunizing measures. The etiologic agent is not yet identified but the virus nature of the disease is rapidly being accepted.

The immunity of the newborn for measles is mostly passive and extends upwards to five months of age, after which an increased susceptibility to the disease begins and continues thru childhood.[13]

Lasting natural immunity is rare and is even denied by some authorities. To the present time the only known method of acquiring lasting immunity is to have the disease in its acute or modified form. Home, of Scotland, attempted to immunize by injection of whole human blood, two hundred years ago.

Passive Immunization of exposed individuals is done largely to modify the disease to the extent of allowing the individual to develop an active "earned" immunity equal to that acquired by an active unmodified attack, yet without the danger of complications so often seen in the unmodified disease. The permanency of such immunity is, however, unknown. In isolated cases it may be advisable, to attempt to entirely prevent the disease, e.g., where the exposed individual is already in a debilitated condition.

Measles modification is carried out by the injection of convalescent serum, adult immune serum, whole blood, and placental extract. Of these methods the use of 5 to 10 cc. of Convalescent Serum intramuscularly five to nine days after exposure or one to two days after the rash has appeared in the contact case, have been most successful in preventing or modifying the disease.

Placental Immune Globulin (Human), introduced in 1933 by McKhann[14] and co-workers, is given intramuscularly in doses of 2 to 10 cc. depending on time interval after exposure, intimacy of exposure and age of the patient.

Results vary from complete failure to modify, prevent, or delay the symptoms; to a modified attack or complete protection.

The modifying action or protective value of adult immune whole Blood or adult immune

serum has proven of little value in either lessening the severity of the disease or in reducing the percentage of common pulmonary complications.

Morales and Mandry[15], at San Juan, Porto Rico in a measles epidemic in June 1928-1929, had an unusual opportunity to test the value of convalescent and adult immune serum. All cases were of familial contact. Working as health officers, with authority, they entered the homes of all cases and exposures. They gave 120 doses of convalescent serum, and 393 doses of immune adult serum to five hundred children under fifteen years of age and over six months of age, using one hundred and eighty-three children in the same families as controls.

Four to six cc. of convalescent serum and ten to forty cc. of adult immune serum was used, reactions occurring in only two of the cases.

Complete protection resulted in 85 percent of those receiving the convalescent serum and in 57.8 percent of those receiving the adult immune serum while 18.6 percent of the controls did not develop the disease. In the attenuated cases there was a moderate temperature rise to 102° F., lasting 24 to 48 hours, the rash appeared one to four days after the injection, showing no erythematous patches and the coryza symptoms were mild. No complications occurred in any of the immunized cases.

Recently an anti-measles vaccine prepared from measles virus grown on fertile hens eggs has been produced by Dr. Geoffrey Rake and Dr. Morris F. Shaffer.[16] Its use at the present time is in process of clinical experimentation.

PERTUSSIS: — Pertussis differs from other contagious diseases of childhood in the fact that 85 to 90 percent of the mortality occurs before the age of three. Under the age of one year the mortality in the non-immunized group may reach 20 to 35 percent. Under the age of five years the mortality rate is greater than that of diphtheria, measles, scarlet fever and poliomyelitis combined. In six consecutive years, the U. S. Public Health Survey, records 42,655 deaths in the United States from this disease alone.

Congenital immunity is probably rare although natural immunity among adults who have never had the disease is more or less common.

There has been an increasing interest and demand by the lay public for protection against this disease. To this end vaccines of various types and dosages have been used for the past decade. Medical literature is crowded with conflicting reports of their efficacy.

Madsen, in 1929[17], and Sauer[18], a short time later, developed and used potent strains of Hemophilus Pertussis Bacillus Phase I, as vaccines in active immunization. The latest Sauer Vaccine is standardized at 15,000 million organisms per cc. and is used as follows: 2 cc., 2 cc. and 3 cc. are injected in alternate arms or 1 cc., 2 cc. and 4 cc. The last dose divided, 2 cc. into each of two previously unused skin areas. An interval of three weeks between doses is recommended. Local and systemic reactions are described as "negligible", particularly after the first two doses.

The eighth month of life is recommended as the proper time to start these immunizations. Used in the early months of life (before the third month) it has produced insufficient antigenic response to protect against the disease.[19]

Double strength vaccines containing 20,000 million per cc. have also been used reducing the number of weekly injections to three, 1 cc., 1.5 cc. and 3 cc. totaling 80-100,000 million pertussis organisms. Opinion differs widely on its usefulness on account of the possibility of increased local reactions. Another commercial laboratory has further concentrated the vaccine to three single weekly doses of 1 cc. containing 20 billion, 40 billion and 60 billion organisms respectively.

Sauer states that immunity develops slowly. Madsen thinks it develops rapidly. Clinical experience has shown that about four months must elapse before anything approaching complete immunity will develop.

Less than a decade ago the total dosage was from three to five billion organisms, now investigators and clinicians feel, that total dosages of from eighty to one hundred and twenty billion are necessary for effective immunization.

There is no practical method of testing the effectiveness of Phase I Pertussis Vaccine, the duration of its protective action or its rate of decline except by clinical observation. Intensity of exposure, type and potency of vaccine, age at immunization and exposure, etc., are all variable factors which act to confuse statistics.

Repeated yearly stimulating doses or additional doses in case of exposure have been recommended. Allergic reactions and foreign protein

sensitization are not to be feared.

Blatt, in 1938,[20] working with a Pertussis Vaccine modified by formalization and used intracutaneously or subcutaneously in total dosage of 30 billion organisms found that this agent proved effective in prophylaxis. Kruegers Endo-antigen, a toxic Filtrate of Phase I Pertussis Bacillus detoxified with formalin, has been used both prophylactically and therapeutically. For prophylaxis 1 cc. then 1.5 cc. are given subcutaneously on alternate days for six doses. It is not yet of proven value.

Pooled Convalescent Serum in 10 to 20 cc. doses intramuscularly in exposed non-immunes has been used. To date the general inavailability of this serum has restricted its use. Available statistics give no definite proof of its value.

Kendrick and Randall, taking adults with a definite history of childhood pertussis, inoculated them with a course of seven to twenty cc. total dosages of Pertussis vaccine over a period of weeks. The blood from these individuals gave a hyper-immune serum of high opsonic index. The difficulties of production and distribution of such a serum makes its general use prohibitive. Immune Rabbit Serum Globulin is still under experimental investigation. Other vaccines, filtrates and endo-antigens have also been used in treatment but their value seems out of proportion to the pain and general discomfort occasioned by the multiple injections required.

POLIOMYELITIS (Infantile Paralysis): — Several agents have been offered and tried in active immunization against poliomyelitis. In 1934, Kolmer[21] introduced a vaccine believed safe and effective for active immunization against the disease. This vaccine contained the living but attentuated virus of poliomyelitis. With the courage of conviction he allowed himself, his assistant and his own children to be given the vaccine, and without harmful effect. Later almost 11,000 subjects were so vaccinated.

Unfavorable criticisms resulting from the occurrence of several fatal cases of the disease developing shortly after the administration of the vaccine halted its further use.

The Park Broder vaccine prepared from killed virus was also discarded as of doubtful value, even though safe. Toomey[22] summarzies the vaccine situation well when he states "Vaccine antigens must be safe, thus they cannot be viable and paradoxically if they are not viable they seem to be of no value."

Bacteriologic studies of the disease by Dr. E. C. Rosenau of the Mayo Foundation have disclosed a pleomorphic green-producing streptococcus, isolated from all of his patients in various stages of poliomyelitis. With this organism he has produced an animal antitoxin which he has used with results which he considers equal to those obtained with pooled adult immune serum. The dosage recommended is 2 cc. for children under two years of age, 10 cc. from two to five years; 15 cc. from five to ten years and 20 cc. for subjects over ten years. These doses to be repeated every 24 hours depending on necessity. Since this is a horse serum its use carries with it all the precautions regarding hypersensitivity and sensitization.

Nasal sprays containing such agents as Zinc Chloride, Tannic Acid, etc., have not proven efficient in prevention and may even exert harmful effects. Pooled Convalescent Serum in doses of 10 to 20 cc. intramuscularly when used under epidemic conditions for passive immunizations gave no corroboration as to its value. Flexner considered it of value in time of epidemic and when given to children exposed to the disease.

This serum is now generally available through the Illinois Health Department Laboratories and is used both for passive immunization and treatment. The case for convalescent serum is however far from convincing. It is administered in 25 to 100 cc. doses intramuscularly or intravenously without fear of local or general reactions. Intrathecally it is not recommended.

In general we may justly conclude that for the present there seems to be no adequate means of specific immunization or treatment either in the control or after the disease has developed.

TYPHOID FEVER: — Typhoid immunization is recommended at eight to twelve years of age or at any time for which occasion may arise. Immunity seldom persists over two years and is never absolute. Typhoid-paratyphoid vaccine is given subcutaneously in doses of 0.5 cc., 1.0 cc. and 1.0 cc. at weekly intervals.

Re-injection of 1 cc. yearly will stimulate the agglutination titer of the already immunized individual.

The routine and recommendations by which these various immunizations may be administered will be subject to the personal opinion of the clinician and the physical condition of the patient. In general the time and method might be as follows:

(1) Vaccinate against pertussis at six to eight months. The Sauer type vaccine may be used in all three strengths, 8 cc. of the 10 billion per cc., 7 cc. of the 15 billion per cc., or 5 cc. of the 20 billion per cc. These total doses to be divided in three separate doses administered preferably at two to three week intervals.

(2) Immunize against Diphtheria between eight and twelve months. The combined Diphtheria-Tetanus Toxoid may be used and seems to be a sensible procedure. Three doses at two week intervals to the amount of 0.5 cc., 1 cc. and 1 cc. seems practical. Schick and re-immunize if and when necessary.

(3) Do a Schick Test three months after Diphtheria immunizations, again at five to six years of age or during epidemic periods and re-immunize if Schick Test is positive. Remember that even vaccinated Schick Negative individuals may sometimes be carriers.

(4) Vaccinate against Smallpox at any age if exposure or necessity requires it, but routinely between three to twelve months. Revaccinate before entering school at six years and again at puberty. Calf lymph vaccine by the multiple puncture method, without shields and during the colder months if possible, is recommended.

(5) Scarlet Fever Toxin in 650; 2,500; 10,000; 30,000 and 100 to 120,000 Skin test doses may be given at weekly intervals followed in one week by a Dick test. Routinely it should not be administered before eighteen months of age and preferably at two or three years.

Under epidemic conditions and in large groups or in child caring institutions its use is more specifically indicated. The duration of this antitoxin immunity is variable and is no guarantee against invasion of the streptococcus Scarlitinae itself.

(6) In co-operation with a nation-wide "Case Finding" effort to eradicate tuberculosis, a Tuberculin Test should be done on children at three years of age and possibly every third year to puberty.

(7) Typhoid Vaccine subcutaneously in doses recommended may be given when and where indicated.

(8) Measles Convalescent Serum in prophylaxis and modification of measles offers good chances of success and its use should be extended. Placental Immune Globulin Human may also be tried. The use of Poliomyelitis Convalescent Serum both prophylactically during epidemics and therapeutically may well be continued until replaced by a more perfect agent.

The chief problem which confronts the physician today, in administering any form of immunizing treatment is not whether he can give absolute or lasting immunity to his patient, but rather, whether he can give sufficient protection to be of practical value. From ten to fifteen percent of failures are to be expected, yet even among these so called failures, the disease may be less severe and have fewer complications. This is frequently the case among those who have failed to obtain complete protection by pertussis vaccine.

Working in this manner, with an ever expanding armamentarium of immunizing agents and always within the bounds of safety, physicians can continue to render valuable aid to their communities in the ultimate elimination of contagious diseases.

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DISCUSSION

Dr. W. L. Crawford, Rockford: Dr. Maurer has summarized well the common practices of immunization against contagious diseases. Little additional information can be brought to your attention but I think that emphasis on a few points from the standpoint of private practice might help crystallize a few things in the minds of younger men and some who have not standardized their office routine procedure.

For fifteen years in immunizing for diphtheria and smallpox, we have followed a routine that has not been necessary to have changed. At first with toxin-antitoxin and later with toxoid we have made it a routine practice to give these diphtheria injections to infants at around six or seven months. We give three injections and never use less than 3 c.c.'s of toxoid. It has been our custom to give a half c.c. at first, 1 c.c. the second injection and 1½ c.c.'s the third time. I know this dosage works. We recheck our children every four or five years and at those ages I know that they develop enough antitoxin to keep the Schick test negative and to maintain it over a number of years.

We vaccinate against smallpox at the same time that we give the second diphtheria injection. This enables us to have an opportunity to see how the vaccination is working when the child gets the third diphtheria injection.

The doctors attitude towards pertussis immunization is changing. We now know that infants in the first months of life do not develop good anti-body protection against pertussis. Drs. Sauer and others have recently reported records and followups of a great many hundreds of children given pertussis immunization in different years and different months and I think the pendulum is now definitely swinging to the administration of pertussis vaccine in the second half of the first year rather than in the first half. I think that in the past our dosage has been too small and it has been increased in recent years with much better protection against pertussis.

Dr. Louis Sauer, Evanston: Based on complement fixation tests and retests on about 800 children of various ages it was found that the earliest age at which the vaccine is likely to confer protection is probably after the seventh months of life. The three-week interval was found to yield a higher incidence of 4-plus complement fixations than the weekly interval between the three injections. We now recommend the three-week interval. It was found that it was not necessary to have an interval of several months between pertussis and diphtheria immunizations.

In 1928, when we began to inject our special H. pertussis vaccine for immunization, Dr. Hektoen advised that we use human blood in the culture medium because it would be safer than animal blood. Without animal protein, sensitization of the infant would also be avoided. From 1934 to date we have had a total of 59 failures (private practice, Evanston Health Department Clinic, and at St. Vincent's). Over 80 per cent of these failures occurred in children injected with vaccine made before 1935. Vaccine made since 1935 has given good protection. The concentration of choice is the new Authorized 15,000 million per cc. vaccine. Additional precautions in its preparation and administration have been added. Local and systemic reactions are less likely to occur when the 15,000 million vaccine is injected at three-week intervals just under the skin of the upper arm in the region of the deltoid insertion. A subcutaneous lump should be seen when the needle is withdrawn. If fever occurs it will respond promptly to aspirin. Nothing is applied locally when a local reaction occurs.

Dr. Maurice L. Blatt, Chicago: In the field of active immunization, those of us who are caring for children have every opportunity to institute these protective measures. The number of adults who consult a physician for such immunization is much smaller, but many parents accept the fact that active protection of their children against infection is desirable.

Anti-pertussis rabbit serum has recently been introduced for research purposes. We are using it for passive immunization. It has given us excellent results so far. We have been able to protect contacts and reduce the severity of the disease in infected children.

One thing which we have neglected is active immunization of both children and adults against tetanus. I want to call your attention to the fact that 24 cases of active tetanus were admitted to the Cook County Hospital during 1939 and 1940. Nineteen per cent of them died in spite of our excellently organized team for the treatment of these cases. Only one of these had had antitoxin prior to admission and only two had consulted a physician because of the injury, resulting in tetanus. It is apparent from these figures alone that only active immunization would protect against tetanus resulting from injuries so slight as not to receive medical attention. In our series, the injuries were so minor that 80 per cent of the patients did not seek medical care. We are in a war at the present moment and in our army tetanus toxoid is not routinely used. It is used by our Naval Academy. In a paper to be published in the Military Surgeon in the near future, I have advocated its use and hope to influence our army to follow the example of the Spanish, English and the Italians who do inoculate their soldiers. We in private practice can do much to reduce the incidence of tetanus by inoculating the children.

I hope that pediatricians will lead in the introduction of tetanus toxoid as they have lead in the use of diphtheria toxoid and other agents for active immunization.

Dr. Archibald L. Hoyne, Chicago: Among the many important procedures discussed in Dr. Maurer's

paper it seems to me one of the most vital in connection with diphtheria is the use of the combined toxoids. I think eventually everyone will be immunizing against diphtheria by means of the diphtheria toxoid plus tetanus toxoid. In this way, one can immunize against two diseases at the same time and in addition the antigenic response is greater to each toxoid when given in combination than when either one is used alone.

In regard to the whooping cough problem, it has seemed to me that the younger infants could be given Dr. Sauer's vaccine before the age of six months; for even though the antigenic response may not be good, possibly some reduction in the degree of susceptibility would result.

We know that most of the infants who die from whooping cough are under one year of age. Consequently, if immunization is not undertaken before the second half year of life the very group which needs protection most does not receive it. For example, during 1938 there were about 10,053 cases of whooping cough reported in Chicago. Among that number there were 66 deaths and all but one of the fatalities occurred either in Municipal Contagious Disease or in Cook County Hospital. By far the larger majority of hospitalized patients were cared for in the former institution, where none was admitted who was more than one year of age. For many weeks the daily average of such patients was approximately 100. Although the general fatality rate was low most of the deaths that took place during the epidemic occurred in Municipal Contagious Disease Hospital, and consequently were among patients under one year of age.

If vaccine were given during the first few months of life I wonder if it might not modify the disease even if it did not prevent it. If such were the case the infant who might ordinarily die, would perhaps survive, and in this way mortality from whooping cough in infancy would be reduced.

Dr. Frederick H. Maurer, Peoria (in closing): In the first place I would like to thank the Doctors for their generous and undeserved discussion.

Dr. Crawford emphasized how different is the situation in administrating immunizing agents in private practice than in regular hospital routine and the necessity of the practical application of immunizations. It is not always possible to have your patients coming in at the intervals at which you want them. You may have to space immunization treatments so that you can give them in connection with your general examinations. Also he mentioned the point of giving sufficient dosage of these agents without producing any harmful effects. That is quite a good point. He specifically advocated the use of 3 c.c. of the toxoid, instead of 2 c.c. He also brought out the point concerning the increased mortality in the younger infants. Dr. Hoyne also emphasized our inability to immunize at the earlier ages, when this protection is most needed.

Dr. Sauer pointed out that the time interval between diphtheria and pertussis immunizations may be short. I routinely do this with my patients.

Dr. Blatt spoke of the rabbit immune serum, which I believe is only in the stage of clinical experimentation. I have also mentioned in this paper, Dr. Blatt's work in 1938, on the use of a filtrate of the pertussis organism in immunization, with which he has obtained some very encouraging results.

The subject of tetanus toxoid immunization in connection with diphtheria is an important one. I think this should be practiced routinely by all physicians but I wish to sound here a note of warning in depending too much upon the protective effect of the toxoid in this procedure. Massive exposure to tetanus infection should have the additional protection of a prophylactic dose of tetanus antitoxin.

Dr. Hoyne spoke of immunizing youngsters against whooping cough before the age of six months because of the possibility of adding somewhat to their protective resources. This is a good idea, since even partial immunity may modify the disease and prevent the commoner pulmonary complications.

I wonder whether, as time goes on, and we immunize all of our potential mothers, we will produce more or less of a "hyperimmunity" in the blood of these mothers which might be passed on to their infants protecting them for five or six months after birth.

THE DORSO-LUMBAR SYNDROME WITH SPECIAL REFERENCE TO REFERRED PAIN

FREDERICK W. SLOBE, M.D., F.A.C.S.
CHICAGO

The frequency with which fibrositis occurs in the mid-back (dorso-lumbar region) is not sufficiently recognized. Such pathology quite commonly gives rise to characteristic referred pain and the combination may well be called the "dorso-lumbar syndrome" to distinguish it from the frequent "sciatic syndrome" associated with low-back involvements.

The literature is replete with articles dealing with low-back pain and the sciatic syndrome but relatively few articles emphasize the frequency of dorso-lumbar involvement. One reason for this, no doubt, is because patients with low-back pathology are usually acutely aware of pain in that region, subsequent sciatic radiation frequently being a secondary manifestation; less often, the sciatic pain appears as the initial complaint, the

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actual back symptoms being either latent or not demonstrable. The reverse situation obtains in the mid-back, instances of prominent referred pain with latent back symptoms or findings being more frequent than instances where the back symptoms predominate. Another reason for the lack of attention paid to the dorso-lumbar syndrome is that sciatic radiation as a symptom-complex does not give rise to many errors in differential diagnosis (though it is often very difficult to establish the exact site of the pathology) whereas the referred pain emanating from the dorso-lumbar region, being less well understood and publicized gives rise to frequent errors in diagnosis. When a pain shoots down the back of the thigh, everyone immediately thinks of sciatica but when the pain is felt in the lower abdomen and groin, attention is often mistakenly focused on various intra-abdominal or other nearby structures as being the seat of the trouble.

It is not within the scope of this paper to dwell upon the pathology in the mid-back responsible for these referred pains. No doubt, some form of fibrositis usually exists — the term "fibrositis" being used for lack of a beter term to include myositis, fascitis, peri-arthritis, arthritis, etc., in fact, any inflammatory or degenerative affection involving the muscles, fascia, ligaments, periosteal insertions, joints, and nerve-sheaths. Any infiltration of these structures may be at fault, whether caused by infection, toxemia, or metabolic disturbance. Usually, the abnormal process is insidious until, with dramatic suddenness, acute pain is experienced. Such acute episodes may indicate an anoxia from vascular spasm, perhaps secondary to local nerve irritation and altered local tissue metabolism. Being often initiated by a strain or twist (sometimes severe, frequently very slight) it appears not unlikely that a torn adhesion or a stretching of tissue whose mobility and elasticity have been diminished by previous inflammatory or toxic infiltration may cause a local nerve irritation which acts as a "trigger" zone. Just as a cartridge waits for the trigger to explode it, so these infiltrated areas, often symptomless before, await some precipitating factor to set off the explosion. This factor is often a strain, sometimes merely a slight movement.

It is logical that the dorso-lumbar region is a frequent site of pathology, because it is a highly mobile part of the spine subject to much stress and strain (the frequent site of compression fracture in this region being ample proof of this). Obviously, the soft tissue structures in this area are subjected to continual stress, especially in those who are physically active. Most of the factors that operate to make the lumbo-sacral region a vulnerable area make the dorso-lumbar area vulnerable also.

The symptomatology is illustrated by the following case reports:

CASE I. — A man, aged 59, while lifting a 200 pound pipe felt a sudden sharp pain in his left lower abdomen, groin, and testicle. He felt weak and walked with difficulty. The pain soon became so severe that he showed symptoms of mild shock. He was sent to the hospital where the examining physician was suspicious of a strangulated hernia because of a soft mass in the left side of the scrotum. Palpation of the scrotum was difficult because of marked tenderness. The mass proved to be an old, small hydrocele. There was tenderness over the left lower abdomen and the skin felt numb when pinched over this area. There was muscle spasm and rigidity of the dorso-lumbar erector-spinae muscles with local tenderness opposite the 1st lumbar vertebra. However, the patient did not complain of pain in his back.

Diagnosis: Referred pain from dorso-lumbar fibrositis, involving the posterior soft tissue structures on the left side. Eucupin in oil was injected paravertebrally around the 12th dorsal and 1st and 2nd lumbar nerves. This caused marked relief of pain, though some tenderness persisted. Of interest is the fact that X-ray examination showed marked spur formation on the lower dorsal vertebrae but relatively little on the lumbar vertebrae.

CASE II. — A hospital orderly, aged 19, on September 23rd, 1940 began to notice pain in his left groin while lifting patients, although no specific strain was recalled. On October 13th, while lifting a patient, he felt severe pain in his left groin which continued through the week. On October 17th while scuffling with fellow-employees, one of them jerked his leg causing an acute increase in the pain, together with slight nausea. Tenderness in the left groin was so marked, palpation was difficult. The examining physician was fearful of a possible small incarcerated hernia. However, the external ring was tight and no hernia was demonstrable. No pain in the back was complained of; however, tenderness was demonstrated over the erector-spinae muscles in the dorso-lumbar region. Impaired sensibility was present over the left lower abdomen over a zone about 3 inches wide above the inguinal ligament.

Diagnosis: Dorso-lumbar fibrositis with referred pain. Local heat and massage afforded relief, the condition disappearing in about three weeks. This young man was a lanky individual with postural im-

balance which apparently caused postural strain with a super-imposed acute episode.

CASE III. — A young man, aged 22, while supporting a heavy bale which started falling from a truck, felt a sudden snap in the extreme lower part of the left side of the abdomen. He said the skin of the abdomen felt as though it were "stretched." He was slightly nauseated three hours later and also developed pain in his left testicle. Additiona lhistory disclosed that his back had been sore at intervals for several months after heavy lifting.

Examination revealed small incomplete hernias on both sides. Coughing did not aggravate the pain. The left testicle was tender. Deep pressure elicited tenderness over the extreme lower part of the left side of the abdomen, the extreme upper and medial part of the upper thigh, and over the upper half of the left buttock. Skin pinching was more acutely felt over these areas than on the opposite side. Tenderness was also present over the left dorso-lumbar region opposite the 12th dorsal to the 3rd lumbar vertebrae and also along the lower margin of the 12th rib. An injection of *2%* procaine into the area of muscle tenderness caused a definite diminution of the pain and tenderness. The patient continued working but as some pain and tenderness persisted, 5 c.c. of eucupin in oil was injected para-vertebrally around the 12th dorsal and 1st lumbar nerves with decided relief. This man had inequality in the length of his legs, the right being ⅜ of an inch longer. Chronic postural strain seemed basically responsible, the added strain of lifting the bale causing an acute exacerbation. This is an instance of hernia being purely co-incidental with no bearing on the complaints, though in such instances often incorrectly so considered.

CASE IV. — A man of 58 slipped while lifting a heavy bag and in trying to keep the bag from falling, felt sharp pains simultaneously in both the left testicle and middle of his back. The left testicle was acutely tender but neither inflamed nor swollen. Beginning bilateral direct hernias were present. Coughing caused no increase in the pain. A local area of tenderness was present over the 1st lumbar spine and the contiguous inter-spinous areas above and below. An injection of *2%* procaine in the tender site around the spinous process caused simultaneous disappearance of both the testicular pain and the back pain in fifteen minutes. This man had a rigid spine from old osteo-arthritis. The local area of fibrositis acted as a "trigger" zone causing referred pain to the testicle. The hernias, again, were purely incidental.

The soft tissues posterior to the spine are supplied by the posterior divisions of the spinal nerves and zones of irritation in that region seem to cause a reflex referred pain down the anterior divisions of the spinal nerves. In the posterior structures of the low-back, radiation extends down the sciatic nerve, whereas in the dorso-lumbar region it most commonly radiates down the 12th dorsal and 1st lumbar nerves.

The sensory distribution of the 12th dorsal and 1st lumbar nerves has been demonstrated by Judovich and Bates to include an area of the lower abdomen three inches wide parallel to the inguinal ligament and a corresponding area of the upper thigh one and one-half inches wide which broadens over the medial upper thigh surface and the scrotum; posteriorly it includes the area bounded medially by the sacro-iliac joint, below by the level of the great trochanter and above by a slanting line well superior to the iliac crest. It should be noted that this zone encroaches upon both the sacro-iliac and lumbo-sacral areas.

The acute pain over the area of reflex distribution is so predominant that most patients make no mention of any recent back complaint. It requires close questioning to elicit an admission of recent soreness in the back and not infrequently there is absolute denial of any such previous trouble. In either event careful examination by digital pressure usually elicits a tender area over the dorso-lumbar region of the involved side, usually from 2.5 to 5 centimeters from the mid-line. More rarely, it may be directly over or between the spinous processes, or even in the tissues caudad to the 12th rib. At times, no tender area exists and here, no doubt, the seat of of the pathology is too deep to be palpated.

At the onset, the pain is often severe, later subsiding to a dull ache as the condition improves. Being a typical reflex, referred pain it cannot be classed as a true neuralgia, though it has some neuralgic characteristics and if the term "sciatic neuralgia" is justified this condition may similarly be termed as neuralgia of the 12th dorsal and 1st lumbar nerves, which are most often involved, though it may include any or all of the nerves from the 10th dorsal to the 3rd lumbar. The trigger zones usually lie in areas of soft tissue irritation in the posterior structures. That the initiating focus may lie in the inter-vertebral articular facets or in the tissues surrounding the nerve-roots cannot be doubted but it is felt that these are less frequent sites.

The question as to whether referred tenderness can occur in the body has been raised. Experience with the above-mentioned patients and other cases would appear to indicate that it occurs

quite commonly. In fact, in addition to referred pain, tenderness almost always co-exists in one or several areas. Tenderness of the testicle, the inguinal canal, the lower abdomen, the upper thigh, and the superior-lateral portion of the buttock usually can be elicited, sometimes largely confined to one area, sometimes to several, and sometimes over all these areas.

This association of severe pain with tenderness causes mistaken diagnoses of incarcerated hernia, appendicitis, renal colic, pelvic disease, etc. causing needless operations to be performed. Two of the above-mentioned patients had small hernias unknown to them since they were entirely symptomless and when such a person develops sudden pain and tenderness in the groin and a small hernia is found, unless a careful examination is made and a detailed history taken, the diagnosis of an acute hernia is frequently made. And, since the referred pain and the tenderness are often self-limited, gradually ameliorating, the mistaken diagnosis may never be rectified.

A diagnostic aid in this condition is the usual, though slight, disturbance of skin or deep sensibility. The sensibility to light touch may be diminished or increased over the lower abdomen or medial, upper thigh as compared with the opposite side. The patient may state that the area feels numb or "different." Pinching the subcutaneous tissues often gives rise to an altered sensation also. At other times only deep muscular tenderness occurs and must be elicited by firm pressure. Similarly, hypalgesia or hyperalgesia to pin-prick may be found. Indeed, these peculiar reflex disturbances have some of the qualities of both neuralgia and radiculitis, yet are not typical of either.

Another procedure which is of diagnostic aid as well as having therapeutic value, often causing immediate relief or diminution of pain, is the injection of a local anesthetic solution. If the tender area in the dorso-lumbar region happens to correspond to a "trigger" zone, the injection of a local anesthetic into that area, frequently causes a marked diminution of the pain and tenderness in the peripheral areas. It would appear that this proves that referred tenderness does occur, since the injection, in such instances, is not a nerve-block of the anterior divisions of the spinal nerves. The reflex arc is simply broken at its source.

Since tender areas over-lying muscular, fascial, and ligamentous structures do not always constitute trigger zones, injections into these areas may be ineffective. Here, para-vertebral injections of the 12th dorsal and 1st lumbar nerves should be effective if the condition is a referred pain along these nerves. From 5 to 10 c.c. of 2% procaine or, still better because of its longer action, 5 c.c. of iodized eucupin in oil are effective. In resistant, recurrent cases a 20% solution of alcohol in 2% procaine may be advisable. When the initial injection is helpful, the later recurrence of pain and tenderness is usually much less severe. A series of injections may be needed. It is not hard to understand the occasional failure of aid by injections since the exact site of pathology is often difficult or impossible to determine. When correctly placed, para-vertebral injections should largely eliminate pain and tenderness over the areas of distribution. Their frequent curative value is difficult to explain. Perhaps the irritating focus, by repeated stimulation, causes a lowered threshold for pain which is elevated toward the normal by the anesthetic solution. When the involved tissue can be reached by direct injection of anesthetic solutions the beneficial effect is often strikingly prolonged. Just why and how this occurs remains somewhat obscure. One factor is the freedom of motion rendered possible by the relief of pain, thus mobilizing stiffened areas. More important, no doubt, is the nullification of disordered nerve function with its associated abnormal tissue metabolism, anoxia, vascular spasm, and altered capillary permeability. When the local nerve elements recover from the effects of the injected solution they may have regained some of their normal functions, thus hastening local tissue repair.

Obviously, treatment must be directed toward the eradication of the cause of the trouble if it can be found. A great variety of therapeutic measures may be needed, including physiotherapy and orthopedic procedures.

To recapitulate, pathology in the dorso-lumbar region is a frequently overlooked cause for referred pain and tenderness in the lower abdomen, groin, upper thigh, buttocks, and low-back region. The distribution of the 12th dorsal and 1st lumbar nerve causes such dorso-lumbar pathology to be confused with intra-abdominal con-

ditions, hernia, testicular complaints, and various affections of the sacro-iliac, lumbo-sacral, and gluteal regions. It is one of the most frequent sites of involvement in the back. In addition to the usual physiotherapeutic and orthopedic measures, and the endeavor to correct the cause, the use of injections either directly into the involved area or by para-vertebral block, is of definite value in many instances.

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DISCUSSION

Dr. C. Paul White, Kewanee: I would like to ask Dr. Slobe a question. He speaks of this fibrositis. He did not definitely diagnose all these cases as fibrositis. We have been hearing a great deal about it.

I would like to ask him what his treatment of these fibrositis cases is and what his prognosis might be in the matter of convalescence.

Dr. D. E. Meier, Kewanee: I would like to ask Dr. Slobe if he can explain the reason for the pain radiating down to the left testicle. I have in mind a number of such cases and wish to coroborate his statements. In those instances that I have in mind I found that they all had hernias. What I would like to ask him is how he accounts for the pain radiating down to the left testicle and so rarely down the right one.

Dr. Frederick W. Slobe, Chicago (closing): Relative to the question as to the treatment of fibrositis, that is a very broad subject and is not within the scope of this paper. Obviously, attention should be directed toward removing the cause, if this can be ascertained. General measures include correction of anaemia, dietetic errors, and metabolic and endocrine disturbances. Physiotherapy and orthopedic treatment are essential.

The injections described, however, frequently have either diagnostic or therapeutic value, or both. If pain is relieved, active mobilization of infiltrated areas becomes possible. Whatever part disturbed nerve function has in these areas may be abolished. Referred pain may cease when the reflex arc is broken. Numbing the highly irritable nerves may enable them to regain a higher threshold for pain. Some patients fail to respond, some are moderately improved, and, in others, dramatic results are obtained.

The left side is affected most often, why I do not know, unless it is because most individuals are right-handed.

STUDIES OF FECES AND CLINICAL CONDITIONS FOLLOWING INGESTION OF GRAPE JUICE

SIDNEY A. PORTIS, M.D.
AND
WM. I. FISHBEIN, M.D.
CHICAGO

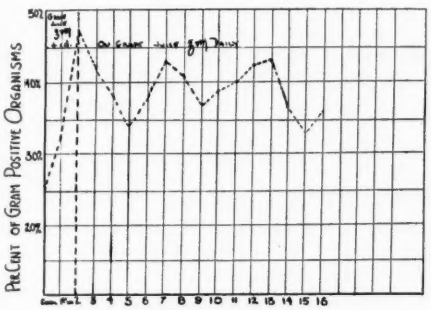
Because of the sugar and mineral content of grape juice, it was felt that this substance might have a marked effect on the intestinal flora. In order to determine this effect, a group of average individuals were selected for study. The bacteriology of their feces before and after taking the grape juice was determined.

It has been known for some time that gram positive organisms may be normal inhabitants of the intestines, and that diet is an important factor in their increase or decrease. The increase in gram positive flora is related to the aciduric content of the stool. Certain substances, such as lactose, as well as a high carbohydrate diet with limitation of protein intake, favor the development of an aciduric flora. If, after determining the average gram positive organism content of the stool it is found that an increase occurs after the administration of certain foods, it is justifiable to conclude that the increase resulted from these additional foods.

The patients studied were on a normal institutional diet which varied but little from day to day. It was found that after taking three glassfuls of grape juice for a period of 10 days, there was a definite increase in the gram positive organisms in the stool.

Method of Study. A group of 50 patients were selected who were free from disease and who were confined to an institution but were engaged in normal daily activities. Routine studies of the stools were made for a period of a week prior to administration of the grape juice. Standard emulsions of the stools were prepared in salt solution. Smears were taken from this emulsion and the number of gram positive organisms seen on the smear were tabulated. After this week of study to determine the average gram positive organism content of the stool, the individuals were given three glassfuls of grape juice daily for a period of 10 days. At the end of this time, the stools were again examined daily and the increase or decrease of the gram positive organisms noted. In addition, the physical characteris-

tics of the stools were also observed, particularly in regard to the consistency. Notes were made on the increase or decrease of the number of stools per day, and whether or not the patients who previously had resorted to laxatives could discontinue their use. Chart 1, series 1, shows that the average amount of gram positive flora in the feces was approximately 25 per cent. prior to the use of grape juice. At the end of two weeks with the grape juice it rose to 47 per cent.



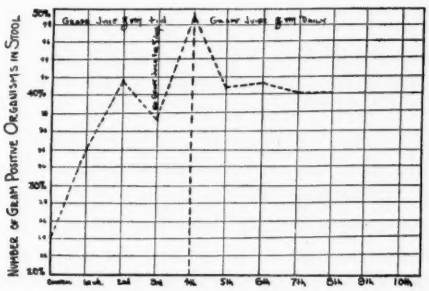
SERIES ONE

To determine whether or not these patients could maintain a gram positive flora on lesser amounts of the grape juice than three glassfuls a day, an eight-ounce glassful daily as a maintenance amount was administered. It was found that when drinking eight ounces of grape juice daily these patients had a slight drop in the aciduric flora, which gradually rose again to approximately 42 per cent. This average was maintained for several months with more or less daily variations. At the end of the period, of about 4 months after the administration of eight ounces of grape juice a day, the number of gram positive organisms remained between 33 and 36 per cent.

It is evident that the amount of gram positive organisms can very definitely be increased by the administration of three eight-ounce glassfuls of grape juice daily. This continued increase can be maintained by the continuance of eight ounces a day. The evaluation of the stools was as accurate as possible by this method. It is to be expected that slight errors within limits would occur in this type of experiment.

A second group of 23 patients were selected for study in which the initial period of administration of three glassfuls of grape juice daily covered four weeks. In this period, five days elapsed at the end of three weeks during which no grape juice was given at all.

It is interesting to note in chart 2, series 2, that the average count before the administration of grape juice was approximately 24 per cent. At the end of the second week, it had risen to 42 per cent. During the period of five days when no grape juice was given, the flora remained at a relatively high level, although it did drop from 42 per cent, to 37 per cent. When grape juice



SERIES TWO

was added again, a definite secondary rise occurred of the gram positive flora to 49 per cent. After attaining this height, it was decided to determine whether or not the gram positive flora could be maintained at this high level in a patient who had established a favorable environment for these organisms when lesser amounts of grape juice were given. When these individuals had eight ounces of grape juice daily, there was a gradual diminution in the number of gram positive organisms in the first week. Then, for the next three weeks it remained at a more or less constant level. In other words, there was a reduction from 49 to 41 per cent, in the first week when eight ounces were given daily but, after that time, for the next three weeks the amount remained at about 40 per cent.

It is evident from chart 2 that in individuals who originally began with a 24 per cent. gram positive flora, the count may be very definitely increased up to 50 per cent, with three glassfuls of grape juice, and a relatively high gram positive flora could be maintained after a suitable environment was established by giving the eight ounces of grape juice daily.

Discussion. From these experiments, it is seen that one can increase the gram positive flora in

stools of normal individuals by the addition of grape juice to the diet. This increase can be maintained and kept up after a suitable environment is formed for the existence of the gram positive organisms.

Few, if any, acidophilus bacilli were found in the stools of the patient before the administration of the grape juice, as determined by stool cultures. After the continued use of the grape juice, however, a very definite increase in the number of acidophilus bacilli was noted on subsequent cultures.

This group of individuals, confined in an institution as they were, under a more or less standard diet, all had a tendency to be somewhat constipated. The majority had to resort to drastic laxatives each night . . . yet not one of them, after the administration of grape juice, had a constipated stool, and none had to use laxatives during the time of this experiment.

Since we know that gram positive organisms are a factor in calcium metabolism, it is possible to postulate that an increase in calcium absorption might occur from the intestinal tract in an aciduric medium. Furthermore, if aciduric flora inhibits the growth of proteolytic organisms, there may be some relation in changing the flora to the prevention of the so-called intestinal toxemia. Whether or not intestinal toxemia is a factor in certain clinical symptoms patients manifest, may be open to debate. There is, however, no question but that persons who have a perfectly easy daily bowel movement have a feeling of well-being, are more free from headache, more energetic and less lethargic, have better appetites, are more alert, and stand physical exertion better than do persons who are habitually constipated and who are depressed because of the infrequency of their bowel movements.

From the observations made in these experiments, the following conclusions are warranted:

1. Individuals normally have a small amount of gram positive bacteria in their fecal content.
2. This gram positive flora can be definitely increased by the administration of eight ounces of grape juice three times a day for a period of two to four weeks.
3. This increase in gram positive flora can be maintained at a relatively high level after the grape juice is decreased to eight ounces a day.
4. Individuals who are habitually constipated are definitely relieved of the constipation after the addition of the grape juice, and find no further need for laxative or changes in the diet other than the use of grape juice daily.

(The grape juice for this experiment was furnished by the Welch Grape Juice Company, Westfield, New York.)

SULFATHIAZOLE IS BENEFICIAL FOR RECURRENT MOUTH DISEASE

It Relieves The Soreness And Promotes Prompt Healing Of The Lesions Involved In Aphthae, Physician Reports

"In 2 cases of recurrent, scarring aphthae [white spots in the mouth, sometimes called thrush, which develop into ulcers], sulfathiazole was beneficial in relieving soreness of the lesions and promoting prompt healing," Richard L. Sutton Jr., M.D., Kansas City, Mo., reports in *The Journal of the American Medical Association* for July 19.

"Sulfathiazole did not prevent recurrences, however," he continues, "but it gave comparable relief a second time. A large dose works better than a small one. I know no other treatment as effectual."

Cases of aphthae, the author says, are not common and are recognized under many names, but all are alike and their cause is unknown.

"Commencing as a small nodule within and beneath the mucous membrane of the lip, cheek or tongue," he explains, "a lesion increases in size, sloughs and causes severe pain, becomes a crateriform ulcer with inflamed, firm borders, runs a course uninfluenced by local treatment and eventually heals, leaving a soft, pliable, depressed, whitish scar. Several lesions may be present simultaneously. The patient, suffering over a period sometimes of many years, can scarcely swallow during exacerbations [increases in severity]. He seeks relief, generally without any success whatever desipte the ingenuity of numerous consultants, and shows a mouth with many scars to the sympathetic practitioner, who knows the better the wider his experience is that he can indeed do little to give relief."

One of the author's 2 patients had had the recurrent ulcers for eighteen and the other 1 for one and a half years. After two months of treat-

ment the first patient said that her mouth healed completely for the first time in four or five years and stayed healed for two months. Then the disease recurred. Further treatment with sulfathiazole caused the lesions to decrease in size, but new lesions continued to appear. The response of the other patient's ulcer to the drug was prompt and gratifying, the ulcer showing good clinical improvement five days after starting the treatment. This patient reported relief from pain within forty-eight hours after starting the drug. The lesion healed satisfactorily but a year later a new sore commenced. Sulfathiazole again produced the same effect of aborting the lesion and giving relief from pain.

TOLD ARMY WILL NOT UNDERTAKE REHABILITATION OF SELECTEES

Commission Advises Government Organize Voluntary Program To Correct Remediable Physical Defects Of Nation's Manpower

The Commission on Physical Rehabilitation, recently appointed by the Health and Medical Committee of the Federal Security Agency, reports in the Medical Preparedness Section of *The Journal of the American Medical Association* for July 19 that it "is authoritatively informed that the Army will maintain its present high physical standards of eligibility for military service and will not undertake the rehabilitation after induction of men classified by Selective Service and Army induction boards as having remediable physical handicaps. In making the following recommendations, the commision has therefore been restricted to a program of voluntary physical rehabilitation.

"In order to provide information concerning the frequency and the nature of remediable defects among men examined for Selective Service it will be necessary to require physicians of Selective Service and Army induction boards to state in every instance whether the physical handicap responsible for classification I B or IV F is remediable in their opinions. Such registrants should be grouped in a special classification and designated as I B-R and IV F-R. Without this information it will be impossible for states and local communities to prepare plans and to estimate the cost of a rehabilitation program.

"The diagnoses recorded on the records of registrants classified in groups I B and IV F by physicians of the Selective Service and Army induction boards give little indication of the possibilities for complete or partial correction. There is a mistaken impression that all defects of registrants in class IV F are not correctable. Actually, registrants in I B have handicaps which restrict their availability to limited forms of military duty, yet the physical disability in some is permanent and non-remediable. Registrants in IV F are suffering from major disabilities which render them incapable of performing any military duties, but some of the men in this category could be restored to group I B or even I A by the correction of their physical handicaps.

"The special classification as I B-R and IV F-R will act as an incentive to voluntary correction of remediable physical handicaps. A comprehensive program for physical rehabilitation of the young men of the nation is important in order to maintain effective man power for industry as well as for military service. It will also have an influence in retarding the development of disabilities of middle age which are responsible for an unemployable group in every community.

"Experiments in voluntary rehabilitation have been proposed for a few local areas by the American Red Cross and several other agencies. A small experiment in a few selected urban and rural communities will not provide a satisfactory yardstick to estimate the cost for the entire country nor can it provide a reliable answer to the problem. The Commission is of the opinion that we must immediately lay the groundwork of comprehensive future plans for physical rehabilitation of our man power by accumulating accurate information in every part of the United States concerning the frequency and the nature of remediable physical disabilities and it therefore submits the following recommendation:

"1. Form 200, 'report of physical examinations,' under the Selective Service and form 221, WD-AGO, 'report of induction of Selective Service man,' should be amended to contain (*a*) a place for the examining physician to state whether or not, in his opinion, the physical defect causing rejection is remediable or correctable; (*b*) the creation of subclassification of I B and IV F (I B-R and IV F-R) to indicate whether or not the registrant is classified as suffering from a remediable or correctable physical defect; (*c*) statements to be signed by the registrant in these subclassifications relative to (1) his willingness to undergo corrective and remedial procedures, (2) permission for release of information pertinent thereto and (3) whether or not he will provide for appropriate treatment out of his own financial resources.

"2. (*a*) Subparagraph *c* of paragraph 165 of the Selective Service regulations should be amended to permit the examination at any time of confidential records pertaining to the physical condition of a registrant by the governor of the state or his designated official representative; (*b*) Subdivision *b* of paragraph 338 of the Selective Service regulations should be amended to provide appropriate parts of the report of physical examination, form 200, in duplicate, and arrangements be made whereby one form may be retained temporarily for rehabilitation purposes by state Selective Service headquarters and state rehabilitation boards.

"3. The following instructions should be issued by the National Director of Selective Service to all local boards: When a local board has finally placed a regis-

trant in class I B-R or IV F-R, the chairman shall write to the registrant advising him of his clasification and of the underlying condition that caused such classification, and shall inform him that the condition is considered remediable by the examining physician. He shall also advise the registrant to have the remediable defect corrected by his own physician or dentist or, if the registrant is unable to bear the necessary costs involved, he may have the work performed by applying to the appropriate agency or hospital in his area. For this purpose the state director of Selective Service shall supply all chairmen of local boards with a list of agencies, hospitals or other institutions located in the neighborhood which are prepared to engage in this rehabilitation work during the period of the national emergency. The chairman of the local board shall further advise the registrant of the time that the board has allowed for the correction of his defect, at the expiration of which time the registrant must present himself to the board for reexamination and reclassification.

"4. In all states and local areas rehabilitation committees should be created jointly by the Federal Security Agency and the Selective Service System to collaborate with professional groups and with local governmental and voluntary agencies in the development of local rehabilitation facilities.

"5. Only a small proportion of the population can afford to pay or will be willing to pay for corrective measures which may make them available for military or industrial service but which do not as yet interfere with their present civilian occupations. Communities differ greatly in the availability of medical facilities, and some states and many local communities will be unable to provide the remedial measures which may be necessary to correct phyical handicaps. Because of national emergency, the responsibility is national as well as local. In order to meet the situation realistically it is recommended that Congress enact legislation to defray the cost. As this is primarily a matter of vital necessity for national defense, the cost should be met directly by federal defense appropriations to the Federal Security Agency, when needed, utilizing fully its available local medical, dental and hospital facilities. Without federal legislation of this nature, it can be predicted that little progress in voluntary physical rehabilitation is to be expected.

"In the opinion of the Commission the alternative to such a voluntary program is lower physical standards of eligibility for Selective Service and compulsory physical rehabilitation after induction into the Army. Action is required along the lines of one or other of these alternatives, for the present standards of physical eligibility have reduced the nation's reservoir of eligible registrants to a number far lower than had been anticipated."

NO RETREAT FOR MEDICINE

The flame-lit skies of bombed British towns can sometimes be seen at night from countries across the Channel. America, too, hears the rumble of the War God's chariot and his hot breath is felt by each of us. This is not strange when every paper in the land carries banner headlines on war and on our defense measures.

Reports are conflicting. The losses of the combatants fluctuate like the stock market and figures are high or low, depending on which side is telling the story. Our thoughts are pulled this way and that by propaganda. It is true that our sympathies lie with the democratic nations, but do not make the mistake of believing that therefore the dictator powers can have no influence over our destiny. Propaganda, we are told, is the most powerful weapon that can be wielded. It sneaks and crawls into the very mind of man or marches in to the tune of martial music. The democratic nations reach us by arousing sympathy; Hitler and his unholy brethren inspire us with terror and try to create the impression that they are invincible. Many are prone to fall under the hypnotic sway of such suggestions and might then assume that a struggle is not even worth while.

Countless old legends are woven around a theme similar to the story of Achilles' heel. It is comforting to remember that there is always a weak point that can be reached, and when poise is substituted for hysteria we know that liberty must eventually conquer.

In a life-and-death struggle such as that now being waged by England, activities must be restricted to the bare necessities. Research goes on feverishly but in the destructive field of war machinery. Medicine goes on too, and we are even told of great discoveries in plastic surgery, wound therapy, and so forth, which evolved during or as a direct result of the last World War.

These too, however, must be considered as children of necessity. The greatest part of medicine has to be laid aside to be dusted off only after the peace treaty has been signed.

In the meantime our American physicians must carry a burden of responsibility not only for themselves but for those of their profession who are homeless and persecuted, for the harassed and overworked in the war zone, for the dead, and for that vast uncounted army of young men who would have become healers if fate had not called upon them to become killers.

We must not permit the fire glow from England to cause panic which will rout the ranks of medicine. We must continue to perform our everyday tasks with as much serenity as possible. We must not discard research as useless in a mad world, for the day will come to aid those who are torn and bleeding so that when the first of destruction dies away, they may lift high the bright flame of liberty that burns undying in their hearts.—*Pennsylvania Medical Journal*, Feb., 1941.

Clinical-Pathological Conferences

J. J. Moore, M.D., Department Editor.

PRESENTATION OF CASES BY
WALTER SCHILLER, M.D., PATHOLOGIST
Cook County Hospital
CHICAGO

NECROTIZING FATTY CIRRHOSIS OF THE LIVER

DR. J. D. KIRSHBAUM.

A. K. a white male aged 65, was admitted to a medical ward October 2, 1940. The examining room diagnosis was cirrhosis with ascites. The patient had noticed a swelling of his abdomen for 2½ weeks. This was associated with jaundice and swollen feet; he admitted drinking three ounces of whiskey daily for the past three years. The rest of his history was irrelevant.

Physical examination: The skin and sclera were icteric. T. 98.2; pulse 100; R. 24; B.P. 128/68. Pupils were equal and reacted to light and accommodation. Heart and lungs showed no unusual findings except for some rales in both lung bases. The abdomen was rounded and distended. A fluid wave could be felt. The superficial veins were dilated. The lower extremities were edematous. The clinical impression was cirrhosis of the liver.

Laboratory: The urine was negative for albumin and sugar and contained much bile. There were a few white blood cells. The urobilinogen was strongly positive. The feces showed chemical blood, and was yellow in color. Blood examination showed Hb: — 54%; RBC: — 2,850,000; WBC: — 5.650. Differential count: — Polys. — 87%; Lymph: — 10%; Mono: — 3%. Kahn test was negative. Culture of the paracentesis fluid was negative.

Blood Chemistry: 10-3-40 — Blood cholesterol, — total 192; icterus index — varied between 50 to 75. Uric acid: — 3.2; the postmortem blood chemistry was as follows: N.P.N. — 140 mg.; Creatinine. — 2.6; Chlorides — 550; sugar 125.

Course: A paracentesis yielded 500 cc., of a greenish fluid and this permitted the liver to be felt four fingers below the right costal margin. He was given supportive treatment. He remained afebrile, lapsed into a coma and expired two weeks after admission about 5 weeks after the onset of his illness.

DR. AARON ARKIN

This patient gave a history of chronic alcoholism, and complained of swelling of the abdomen and jaundice for about three weeks. A diagnosis of cirrhosis was made. In such a case the inspection of the patient is often suggestive of so-called alcoholic cirrhosis. First, about eighty percent have a pectoral alopecia. Second, we often see small spider telangiectases on the face, neck, shoulders, or chest wall. The veins of the abdominal wall are often dilated and the blood flow is from below upward. Very often we find hemorrhoids. Palpation reveals an enlarged liver in about three fourths of the cases. The enlargement is often due to marked fatty infiltration as well as liver cell regeneration and scar tissue formation. Especially important in differential diagnosis is the presence of a palpable spleen. Such a finding in a case with jaundice speaks for cirrhosis and against an obstructive jaundice. A very frequent symptom of cirrhosis is hematemesis or melena from bleeding varices of the esophagus or stomach. This may be the first symptom of cirrhosis. It occurs in about twenty percent of cases. I have often visualized the varices in the esophagus with barium. I have also noted the presence of edema of the feet in early cases, easily mistaken for a beginning cardiac decompensation. I believe this to be due to some pressure on the iliac veins

from fluid already present in the pelvis. Another significant finding in cirrhosis is the macrocytic anemia, probably related to the chronic gastritis and liver changes with disturbance in the formation and storage of the anti-anemic factor. The urine usually reveals an increase in urobilinogen. There is often a reduction of the serum albumen-globulin ratio. In closing I wish to emphasize the importance of an early diagnosis of cirrhosis in the pre-ascitic stage, in the hope of preventing the fatal ascitic stage. Any patient with a history of chronic alcoholism and a palpably enlarged liver, especially when there is a pectoral alopecia, should be diagnosed as an early cirrhosis. A positive bromsulphalein test will confirm the diagnosis. Alcohol and spices should be prohibited. Jaundice occurs in about forty percent of cases of cirrhosis. It may be mild or severe. When marked it indicates severe liver cell necrosis, which may lead to a fatal hepatic coma with icteric nephrosis and azotemia.

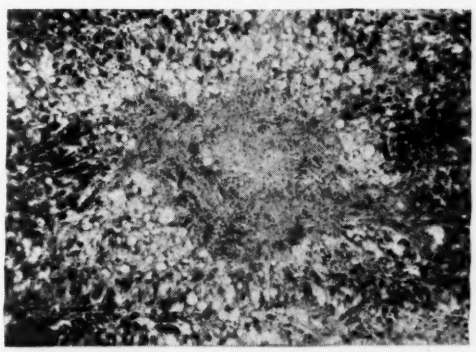
DR. WALTER SCHILLER.

The body is that of a well developed, well nourished, slightly obese, white male with marked yellow discoloration of the skin and conjunctiva. There is no pitting edema. The abdomen is markedly distended and fluctuant. The thorax is barrel-shaped. The midline fat measures up to 18 mm., in thickness and the abdominal cavity contains more than one (1) liter of a pale yellow slightly turbid fluid. The heart reveals nothing of pathological importance and the aorta, except for a moderate amount of fatty degeneration of its intima, is essentially unchanged. The lungs reveal evidence of a chronic emphysema; they have a feathery consistency and their pleural surface is uneven due to numerous bullous projections. On transverse section the lungs are dark-purple red, almost black, in color and on pressure large quantities of a slightly hemorrhagic, yellowish serous fluid escape. The esophagus presents focal thickening and focal desquamation of the epithelium. Distended veins can be seen in the lower one-third of the esophagus. The mucous membrane of the stomach is pale gray in color and is so thin as to be transparent and reveals the distended vessels of the underlying submucosa (atrophic chronic gastritis). The spleen weighs 410 gms., and is extremely soft. The thin gray capsule shows only indistinct markings. On transverse cut the half-fluid splenic parenchyma projects over the surface. The liver weighs 2335 grams and has only a moderately firm consistency. The thin, perfectly smooth capsule is adherent to the inferior aspect of the diaphragm on the right side. On section the liver tissue is greenish-gray in color with small red dendritic markings. The gall bladder contains 30 cc., of a thin greenish bile. All of the large bile ducts are patent. The pancreas weighs 250 grams and on section presents a few dull grayish yellow acini of glandular tissue which are imbedded in pale gray fat tissue. A few bright sulphur-colored spots of fat necrosis are scattered over this fat tissue. The kidneys are moderately firm. The capsule strips with ease from the smooth pale purple surface. On transverse cut a yellow discoloration of the parenchyma becomes evident with its distinct markings. The contents of the intestines reveal the presence of bile pigments; those in the small intestines being green in color and those in the large intestines being brown in color. Microscopically the liver is composed of small and large pseudo-acini which do not have the radial structure of normal acini. They are separated by small, slightly increased periportal fields. The intra-acinar connective tissue which connects the peri-portal triades similarly presents only a minimum increase. There are dense lymphocytic infiltrations in the triads and a few regenerating and proliferating bile ducts. The liver cells present a high degree of fatty degeneration and in some areas, intra-and intercellular bile casts. There are numerous small foci of liver cell necrosis.

DISCUSSION

It may seem questionable whether we are correct in classifying a case like this as one of cirrhosis. This name was coined by the first investigators for an induration and fibrosis of the liver. The Greek word from which this name (cirrhosis) is derived is the same as that from which scirrhous carcinoma is derived and the latter means a carcinoma of high resistance due to the increased amount of connective tissue. Now-a-days we know that in the degenerative processes which involve the liver in some cases of chronic alcoholism and more acutely in cases of intoxication by spices and similar irritating substances, the fibrotic induration is only one component of the pathological changes which occur. Degeneration, (usually of the fatty type, but occasionally of the necrobotic type), of the liver cells is the second and even more important component of the changes which

occur. For the latter there is a compensatory regenerative bile duct proliferation. In the classical Laennec Hypertrophic Periportal Cirrhosis the fibroplastic component is in the foreground but in many cases we see the degenerative and the necrobiotic components play the cardinal role. Apparently not only the type of toxic substance but also the constitution of the patient is of importance as to which component will play the major role. Certain individuals have a general constitutional tendency for a strong connective tissue proliferation response. Such individuals develop keloids after injuries which result in only thin linear scars in the normal individual. A similar constitutional fibroplastic response may be responsible for the development of a hypertrophic granular cirrhosis, whereas in individuals without such a tendency the degenerative component is the predominating one. However, it seems justifiable to use the name cirrhosis for the combination of degeneration, fibrosis, and regeneration of the liver, provided we characterize the individual cases by adding the proper adjectives to describe the predominating findings. Thus our case is one of necrotizing, fatty cirrhosis. The rather frequent combination of chronic bronchitis with liver cirrhosis deserves some explanation. The veins of the bronchial mucosa discharge their blood anteriorly into the Vv.anonym., the V.cava superior and in the Vv. pulmonales, and posteriorly (this seems to be of greater importance) into the V.azygos. This vein communicates with the V. lumbalis ascendens, and the ascendens with the V. iliolumbalis. The V. iliolumbalis anastomoses with the Plexus Sacralis, at the anterior aspect of the sacrum, and this plexus with the Plexus Hemorrhoidalis, behind the rectum. The Plex. Hemorrh. gets blood from the middle V. hemorrhoidalis and this vein anastomoses with the inferior V. mesenterica, a tributary of the Portal Vein. By this way a collateral circulation is established: v.mesent. inf.- v. hemorrh. media- plex. hemorrh.- plex. sacr.- v. iliolumb.- v. lumb. ascend.- v. azygos- c. cava sup.- by this collateral way venous blood from the intestines can reach the V. cava sup. without passing the liver through the portal vein if the passage is impaired by cirrhosis. On the other hand the V. coronaria ventriculi. of the stomach, a tributary of the portal vein, anastomoses along the cardia with the esophageal veins, which empty in the v. hemiazygos, which opens into the V. azygos- a second collateral from the portal to the cava superior which contributes to the engorgement of the V. azygos. In our case, at autopsy, the V. azygos was found to be markedly congested and was thicker than the little finger. This congestion extended back to the posterior bronchial veins and provoked a passive congestion and a chronic edema of the bronchial mucosa and hypersecretion of the glands thus causing a chronic catarrhal bronchitis. The etiological mechanism of this duplicates that of the stasis catarrh of the stomach in heart failure. The lipomatosis or steatosis of the pancreas as seen in this case and the causative factors thereof have been investigated. In some cases it appears to be a chronic degenerative process caused by chronic inflamation. The slides of our case present a moderate lymphatic infiltration of the septa. The increase of the fat tissue may be a simple exvacuo proliferation as is generally carried out by the interstitial fibrous tissue. Concerning the laboratory findings, the highly positive urobilinogen may be explained by the degeneration of the liver. The liver parenchyma under normal conditions transforms the urobilinogen, which is formed in the intestines as a result of the reduction of bilirubin by the colon bacillus (B. coli), back into bilirubin and excretes it with the bile. The degenerated liver cells are not capable of carrying out this transformation and the urobilinogen escapes into the blood stream. The high N.P.N. is to be traced back on the one hand to the destruction of protein in the liver necrosis and on the other hand, to the degenerative changes of the renal tubules caused by the jaundice. The damaged epithelial cells, instead of shifting only the water out of the glomerular secretion toward the interstitial capillaries and thus concentrating the urine, allow the N.P.N. which has already been excreted by the glomeruli escape with the water back into the blood stream.



Liver:

In the center a focus of complete necrosis with lymphocytic infiltration surrounded by a zone of fatty degeneration. In the periphery only to the right and to the left strands of normal liver cells can be seen.

Anatomical Diagnosis:

Necrotizing fatty cirrhosis of the liver with non-obstructive jaundice,

Progressive steatosis of the pancreas and foci of fat necrosis,

Chronic catarrhal bronchitis and edema of the lungs,

Varicose esophageal veins,

Infectious hyperplasia of a cirrhotically enlarged spleen,

Chronic atrophic gastritis,

Moderate dilatation and severe myo-degeneration of the heart,

Moderate coronary sclerosis,

Icteric nephrosis of the kidney,

Hemorrhagic trigonitis of the urinary bladder,

Small polyp of the rectum,

General adiposity.

ACUTE AND CHRONIC GLOMERULONEPHRITIS

DR. J. D. KIRSHBAUM.

Patient was a 17 year old, white boy, who was admitted to the Cook County Hospital on the 27th of October 1940, with an examining room diagnosis of Pulmonary Pathology. He was well until two weeks prior to admission when he started having vomiting spells which was followed by pain in the right upper abdomen. This was followed by a sore throat, pain in the left chest and precardial region. For three days he noticed shortness of breath. On admission physical examination disclosed a pale looking boy. The throat was injected. Temperature was 100.4; the pulse rate 96; and respirations 26. The blood pressure was 190/129. There was dullness with increased tactile and vocal fremitus, and rales in the left lower lung field. The clinical impression was chronic glomerulonephritis.

Laboratory: *Blood*: Hb: — 36% R.B.C.: — 2,190,000; W.B.C.: — 9,950; differential smear showed 87% polymorphonuclear leucocytes; 11% lymphocytes; and 2% basophiles. Non-protein-nitrogen was 280 mg., creatinine 20,0 mg. Blood chlorides 440 mg. CO_2 combining power 28 vol. percent. Serology was negative. The urine showed 4 plus albumin; sp. gr. 1.010; and red blood cells microscopically. X-ray examination of the chest was negative. In the ward the patient had difficulty in urinating. In spite of parenteral fluids the urinary output was scanty. He became markedly dyspneic, semi-stuporous and expired 11-3-40, on the 7th hospital day or 3 weeks after the onset of his illness.

DR. WALTER SCHILLER.

On autopsy, we found a well-developed, well-nourished, white male. The skin is white and markedly pale. No edema can be found. Examination of the external genitalia reveal a slight infantile hypoplasia especially of the testicles. Over the skin of the chest tiny white crystals can be seen. On opening the peritoneal cavity, the intestinal loops are slightly distended by gas.

The peritoneal serosa is smooth, and shiny, and the peritoneal cavity free. The pericardial sac contains 20 cc., of a turbid hemorrhagic fluid from which red blood cells have sedimented. The epicardium is soft, and appears to be slightly thickened. The heart, 320 gms., is moderately contracted and shows a fibrous epicardial plaque, on the anterior aspect of the right ventricle close to the apex. The myocardium is pale, pink-brown, and firm. The valves are all normal. The coronaries present moderate fatty degeneration of the intima. The wall of the right ventricle is slightly thickened, and the trabeculae carnae are plump. The pleuras are free. The lungs, particularly the lower lobes, show on transverse cut a few dark consolidated areas of bronchio-pneumonia. The mucous membrane of the bronchi is pinkish-brown, and the lumen contains frothy mucinous fluid. Hilar and tracheal lymph nodes are markedly enlarged, soft, grey and edematous. The liver, 1500 gms., is moderately firm. The spleen, 145 gms., is rather soft and on section the dry surface reveals large and very distinct lymph follicles. The kidneys, 150 gms., are small and moderately firm. The capsule strips with moderate ease, leaving a slightly granulated light yellow, white surface, with numerous pin point size dark purple red spots. On section the cortex is markedly reduced in size to 3 to 4 mm., the markings and the demarcation between the cortex and the pyramids are indistinct. The mucous membrane of the renal pelvis is normal. The urinary bladder contains about 100 cc., of a pale, yellow, thick turbid, almost mucinous urine. The wall is markedly thickened by edema. The mucous membrane is light grey with congested vessels and small petechiae in the region of the trigonum.

Microscopical examination: The kidneys present different types of changes in the glomeruli. The vast majority show marked proliferation of the epithelial and endothelial nuclei of the tufts, which contain numerous leucocytes in the lumen. The endothelium of the parietal layer of Bowman's capsule, showed a marked proliferation by which it fills Bowman's space almost completely. The connective tissue of the capsule

presented a moderate thickening only. There is another smaller group of glomeruli with slight proliferation of the tufts which are rounded up and present no indentation of the peripheral contour. Here Bowman's space is free, the endothelium is normal but the fibrous part of the capsule is markedly thickened. A third group of glomeruli shows more or less complete hyalinization. The first group represents acute and subacute changes, the second and the third chronic changes. The tubuli of the chronic group are involuted. The tubuli of the acute group are dilated and filled with proteinic debris. Some of them present a marked dilatation and are filled particularly in the subcapsular region, with red blood corpuscles. These distended tubuli which are stuffed with red blood cells, correspond to the red spots seen on the surface on gross examination. In the lower part of the cortex and in the pyramids there are numerous radial areas of dense leucocytic infiltration, ascending along the canaliculi from the pyramids to the cortex, giving evidence of an old pyelonephritis. The vessels are almost unchanged. The fat stain shows fatty degeneration of the tubular epithelium in the inflammatory areas. The lungs present numerous areas of alveoli stuffed with leucocytes. The smaller bronchi and bronchioli are filled with pus cells and the mucosa shows inflammatory infiltration. In many areas alveoli are fused together by emphysematous deficiency of separating walls.

DISCUSSION

The history of this case gives evidence of an old chronic process on which an acute one was superimposed. The old represents a chronic nephritis, which has caused the hypertrophy of the left ventricle by increased blood pressure and the granulation and shrinkage of the kidneys. The cause and onset probably was some infection in early childhood. The history gives no information. This happens frequently since the first phases of many cases of nephritis are so insidious and inconspicuous on clinical observation. A few weeks ago the patient developed an acute sore throat of a septic type. This provoked an acute reaction of the kidneys which already were sensitized by the chronic process. These two strata are manifested in the kidney by the two types of changes in the glomeruli. The fibrosis, and hyalinization, representing the chronic, and the endothelial proliferation representing the acute and subacute renal reaction. The latter evidently started a few weeks before death beginning with a hemorrhagic glomerulitis. At death there were no longer any red blood cells in the glomerular space. They had already been passed into the dilated tubuli where they produced the flea bite like red spots as gross inspection. A third pathological change is due to the ascending pyelonephritis which may have contributed to the sensitization and undoubtedly has contributed to the final renal insufficiency and uremia. Cases like these have to be classified as glomerular diseases in the classification of renal affections of Bell, who distinguishes glomerular, vascular, tubular, and interstitial changes. Whereas in former times and particularly at the time when Volhard and Fahr laid in the foundation of kidney pathology, glomerular affections where in the foreground and the vascular sclerosis was only a small annex of kidney pathology, now a days and with us the true glomerulonephritis has become rare compared with the ever-increasing incidence of vascular nephrosclerosis. We see in our material at the morgue about ten times as many cases of nephrosclerosis as we do of true gomoerulonephritis. The hypertrophy of the left ventricle goes with chronic nephritis which cannot explain the hypertrophy of the right ventricle. This has to be traced back to the emphysema which evokes a stronger reaction in the right heart of a child than in that of an adult.

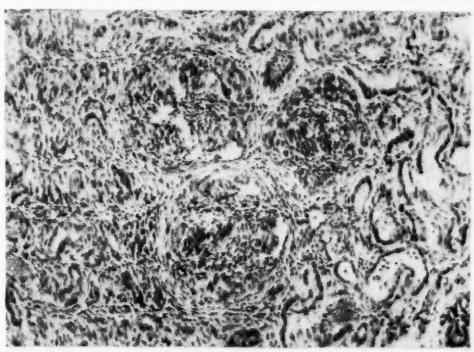


Fig. I. Three glomeruli with "crescents" (acute proliferation of Bowman's capsule endothelium).

Anatomical diagnosis:

Subacute hemorrhagic nephritis superimposed on chronic glomerulonephritis, chronic cystitis with edema of the bladder, bronchopneumonia of both lower lobes, slight hypertrophy of the left and marked hypertrophy of the right ventricle, acute hemorrhagic pericarditis, infectious hyperplasia of the spleen.

CARCINOMA OF THE CECUM, and CARCINOMA OF THE RECTUM

DR. J. D. KIRSHBAUM.

Patient was a white male aged 54, who was admitted to the hospital September 21, 1940. He stated that during the past year he had lost twenty pounds in weight, had abdominal pains,

distension and a progressive weakness. His appetite was good, but did not eat because of abdominal discomfort and vomiting which resulted. An appendectomy was performed in December 1939, at which time a tumor was found. Two months later he was admitted to the Billings' Hospital complaining of colicky pain in the lower quadrant, and vomiting. Examination disclosed a tumor mass in the region of the caecum, about the size of a fist. On February 21, 1940, he had a resection of the caecum, adjacent lymph nodes, and three and one-half feet of ileum. An ileocolostomy was performed. X-Ray examination of the intestinal tract revealed an annular carcinoma of the cecum. He made an uneventful recovery, and was given nineteen x-ray treatments during the next nine months.

Physical examination: Disclosed a marked distention of the abdomen. It was tympanitic. A huge, firm, irregular mass in the liver area was felt. A right rectus surgical scar was present. Temperature 98.6; pulse 100; respiration 28. The prostate was small. A firm mass was felt in the anterior wall of the rectum, 10 cm. from the anus which was considered to be extrinsic.

Laboratory: Urine- albumin-trace; bile $^{+++}$; occasional white blood cells. Urobilin $^{++++}$.

Blood — 9-23-40: — Hb 60%; RBC 3,300,000; WBC 7,850; Polys 91%; Mono 7%; Lymph 2%

9-23-40: — NPN 50; Uric acid 5; icteric index 15; Blood Kahn-negative.

Course: The abdomen remained distended, he was given fluids and blood transfusions, but gradually went downhill and expired one week after admission.

Impression: Metastases to the abdomen from a carcinoma of the cecum (previously removed).

DR. L. SLOAN.

This case presents several interesting features. We are told that previous to the original operation this patient had abdominal pain, distention, marked anemia and alteration in bowel habit. The history given by the patient on his recent admission to the Cook County Hospital might well have been the history given originally. You will note the chief complaint is distention and distress produced by eating. The patient's actual appetite remained very good but he did not eat because immediately after there was produced in the lower abdomen a distress which was sufficiently severe to prevent enjoyment of his food. This is very suggestive of colon pathology. You will recall that with acute colitis the common complaint is abdominal distress initiated by or shortly after the ingestion of food. The patient's appetite remaining so good we are clinically prompted to believe that there is no pathology in the stomach proper. The original marked anemia again suggests a papillary or fungating lesion of the colon which by chronic bleeding produces anemia. On examination of the patient previous to entrance into this hospital a mass was found in the pelvis which was regarded as extrinsic. Examination by a resident here disclosed a mass which could be felt on rectal examination but which was regarded as again extrinsic. In other words this mass was regarded as a drop metastasis in the pelvis from a previous upper abdominal malignant lesion. The history and physical findings warrant us in a clinical diagnosis of fungating adenocarcinoma of the cecum which was resected and from which the patient made a very excellent mechanical recovery. We must add thereto the diagnosis of metastatic carcinoma involving the liver and undoubtedly most of the structures in the abdominal cavity. Of particular interest is this mass in the pelvis. Is this a primary carcinoma of the rectum from which metastases have spread to the liver and other organs of the abdominal cavity or are the metastases the result of a primary fungating carcinoma of the right colon? The absence of metastases in the liver and other structures at the time of operation would suggest to us that we have been dealing with two independent carcinomas, one in the right colon and now a second one primarily in the rectum. The clinical interest rests in the history of a good appetite, failure to eat because of distress in the lower abdomen, marked anemia preceding the original operation, recovery from operation, the rapid development of extensive metastatic growths throughout the abdomen, and a mass which was palpable by rectum. Is this a drop metastasis or a new growth? One would incline to the diagnosis of two separate malignant growths.

DR. W. SCHILLER.

The body is that of a poorly nourished white, emaciated male. The skin shows a pale yellow,

waxy discoloration. There is a tatoo mark on the right and one on the left forearm. The abdominal cavity contains about 2500 cc. of a turbid reddish brown fluid. The omentum is rolled up and indurated, and adherent to the anterior abdominal wall, at the site of an ancient 5 cm. right rectus surgical scar. The parietal serosa of the intestines all over is covered with numerous, firm, grey pin head size nodules. The pleura presents a few fibrous adhesions particularly over the right upper lobe. The lungs are crepitant and on transverse cut are very moist. The heart weighing 290 gms., has a firm, purple brown myocardium. The valves are all normal. The stomach is distended and contains hemorrhagic mucoid fluid. The mucosa is dirty reddish brown, smooth and in the prepyloric region is studded with small purple red spots. The spleen weighing 250 gms., is soft, dark purple pink, with indistinct markings on transverse cut. The liver weighing 3265 gms., is very firm, and the surface is studded with pin head to 15 mm., flat, greyish white nodes, some, particularly the larger ones, are slightly umbilicated. On section the surface is bright yellow green, mottled with large greyish white, sharply demarcated nodes. Some of branches of the portal vein are occluded by firmly adherent, soft, gray tumor tissue. Corresponding to the area supplied by one large obstructed vein there is a triangular light purple red, sharply demarcated infarcted area 8 cm. long which can be seen in the subcapsular region of the right lobe. The gall-bladder and pancreas present no pathology. The terminal ileum and cecum are missing, and there is a side-to-side anastomosis, in which region the bowel is kinked and firmly adherent to a mass in the mesentery measuring 3 cm. in diameter. The small intestine contains thick reddish-brown fluid, and the colon contains soft, black fecal masses. The cortext of the left adrenal is partially replaced by grayish-white tumor tissue. The kidneys weighing 370 gms., have a finely granular pale purple brown surface. Bladder and prostate are normal. The prostatic veins contain numerous phleboliths. In the rectum, 9 cm., from the anus there is a circular slightly elevated flat tumor plaque, measuring 45 x 26 x 7 mm.

Microscopic: The node in the rectum presents carcinomatous transformation of a group of glands in their normal localization. These glands meet with the normal glands in a sharply demarcated cleavage line without intermedial transition. From these carcinomatous glands small clusters and single cells in great numbers invade the submucous tissue. Whereas the superficial glandular carcinoma presents a high maturity, the invading part is completely dedifferentiated and characterized by many atypical bizarrely shaped cells. In the omentum, the adrenal, and the liver as well as in the perirectal tissue the carcinoma, partially duplicates the dedifferentiated single cell type of carcinoma, forming alveolar structures lined by low columnar epithelium, and filed with a mucin secretion in which desquamated swollen and necrobiotic carcinoma cells can be seen floating. The superficial development and the characteristic high maturity proves the rectal node as a primary. The case consequently, must be classified as a double carcinoma, first of the cecum, then of the rectum. Whether the two carcinomas existed together and simultaneously or whether the rectal carcinoma developed only after the removal of the cecal carcinoma is difficult to decide. The small size of the rectal carcinoma makes it likely that it developed recently i.e. after the removal of the cecal carcinoma. It is a traditional experience that a rectal carcinoma takes a half year to become semi-circular and one year to become circular. Following this experience we would date the origin of the rectal carcinoma 4-5 months back. This suggestion is supported by the negative findings on clinical, rectal, and proctological examination. It is possible that the carcinoma was preceeded by a polyp which later became malignant, but there was no way to prove this at the autopsy.

In double carcinomas we distinguish two groups: that group in which both carcinomas develop in the same organ, and the other group in which the carcinomas develop in two different organs. These groups are then subdivided as to the time of their development, i. e. whether the two carcinomas developed simultaneously or after each other successively. The double carcinoma deserves a special interest since it throws light to the etiology of carcinomas in general.

In case that carcinomas develop on a generalized constitutional tendency, double or multiple carcinomas should be found frequently, provided that the constitutional tendency can not be changed by the presence of a carcinomatous focus in the

body. Hansemann about sixty years ago pointed out that multiple and repeated carcinomas would be found in a much higher frequency if constitutional tendency were the deciding etiological factor. On the other hand, in the middle of the 19th century Beneke established the conception of frequent incidence of what he called a "tumor race," founded on his observations of different families in which numerous members produced several partially benign, partially malignant tumors. These antagonistic interpretations lead to a second problem: whether the constitutional tendency for the production of malignancies if and where it exists is a general one or confined to one organ. Under different conditions both possibilities may exist. An illuminating but only partial answer to the question has been given by Clara Lynch in a very striking experiment. Using two strains of mice, one in which thru cross breeding by the method devised by Maud Slye, a high incidence of spontaneous carcinoma of the breast was found, and a second strain with almost no spontaneous malignancies, she exposed both to identical tar painting of the skin. Both strains produced skin carcinomas at the same time with the same incidence and of the same type. This experiment proves that at least with such strains of mice the hereditary tendency for carcinoma is not a general one but a local one fixed to the breast tissue.

Some observation, from the human pathology leads to a similar conclusion. The carcinoma of the uterine cervix is one of the most frequent of all human carcinomas, ranging generally as the third most frequent carcinoma (the carcinoma of the breast and stomach, being first and second). By clinical experience it has been found that after amputation of the cervix, the new cervix, it means, the new external os, which is several millimeters higher than the physiological os in most of the cases stays unchanged. In some cases which are not too rare inflammatory erosions and ulcerations develop like the physiological cervix. Even when we don't accept the theory that inflammation and erosion play a great role in cervical carcinogenesis, a theory which seems not to be justified, — we would expect* a certain incidence of carcinoma after the amputation of the physiological cervix in the new cervix. As far as I have been able to check in the literature, in spite of the many thousands of amputations of the cervix carried out all over the earth, only 3 or 4 carcinomas of the new cervix at the new external os have been observed. This discrepancy can only be explained by accepting the theory that the tendency of carcinoma is locally limited to the physiological external os.

A very interesting group of double carcinomas are the double carcinomas of symmetrical organs as Fallopian tubes or female breasts. When we see that the carcinoma of the tube, in about 70% of all cases, originates from primary foci in both tubes, which usually present no evidence of a previous high grade inflammation — whereas the overwhelming majority of severe salpingitis cases develops no carcinoma — we have to discard the importance of chronic inflammation and irritation as causative factors for carcinoma of the tube. The experience of double carcinoma in symmetrical organs is duplicated by the numerous cases of identical twins, who developed at the same time and in the same organ the same type of malignancy, — observations which stress the Constitutional factor in carcinogenesis. Our case represents two probably simultaneous carcinomas or possibly carcinomas of the successive type with a very short interval of time elapsing between their onsets. From the embryological and histological point of view, the rectum, and the cecum are related so closely together that we may classify the two carcinomas as developing from one organ. The question whether the metastases were produced by the cecal, or by the rectal carcinoma cannot be settled by histological or cytological criteria since the carcinomas of these two organs are too similar to permit safe differentiation. However, the complete analogy of the type of carcinoma cells in the rectal submucosa and most of the deposits, as well as, the clinical history justifies the conclusion that the metastases are due to the rectal carcinoma. Generally the carcinoma of the rectum in its expansion for a long time is limited to the perirectal tissue and the pelvic lymph nodes. At least for the first few months spread to the liver is rare. Anatomically it may be explained by the anatomosis between the rectal veins, the hemorrhoidal plexus, and the inferior mesenterial vein which opens into the portal vein. The quick spread and extensive metastasing of the rectal carcinoma suggests a diminished resistance or a sensitization thru the previous cecal carcinoma.

*Corresponding to the similarity of anatomical and physiological conditions.

News of the State

PERSONALS · COMING EVENTS MARRIAGES · DEATHS

Benjamin Goldberg, President of the American College of Chest Physicians addressed the Indiana State Medical Society at Indianapolis on September 24, subject "Organized Medicine and Tuberculosis."

A joint meeting of the Greene and Jersey County Medical and Dental Societies was held in White Hall on September 12th. Dr. Carroll W. Stuart of Chicago gave a lecture on "Surgery of the Head and Neck" calling attention to many interesting and unusual conditions that may develop in the region of the head and neck, some of which may originate in other organs. He emphasized the importance of careful diagnostic procedures before extraction of teeth in doubtful cases.

A paper on "The Prevention of Blindness Among Babies in Illinois," prepared by the Society for the Prevention of Blindness was read. The physicians of Greene and Jersey Counties are to be congratulated on their record as shown by the statistics for these two counties.

The Eye and Ear Research Fund, Inc., announces a two-day course in practical gonioscopy which will be given at the Illinois Charitable Eye and Ear Infirmary beginning Friday, October 24th, at 2 p. m., and finishing on Sunday, October 26th, at noon. Applications and requests for particulars should be addressed to the Dean of Instruction, 904 W. Adams St., Chicago, Illinois.

James H. Hutton was invited to address the Terre Haute Academy of Medicine at Terre Haute, Indiana on "Practical Points in Endocrine Diagnosis and Therapy," October 3rd.

COMING MEETINGS

October 14 — Effingham County — Benwood Hotel, Effingham — 6:30 P. M. — Dr. Lewis T. Gregory of Urbana — "Gynecological Problems and Their Treatment"

October 14 — Warren County — Hawcock's Cafe, Monmouth — 6:00 P. M. War Medicine program

October 14 — Bureau County — Perry Memorial Hospital, Princeton — 6:30 — John S. Coulter, M. D. — "Home Treatment of Chronic Arthritis with Physical Agents"

October 16 — Post Graduate Conference, Lincoln

October 23 — Post Graduate Conference, Moline

October 30 — La Salle County — Starved Rock Park Lodge, Starved Rock — 6:30 — James T. Case, M. D. — "Some Interesting Phases of Gastro-Intestinal Diagnosis"

November 6 — 8th District Post-Graduate Conference, Danville

November 6 — Southern Illinois Medical Society

November 11 — Kankakee County Medical Society — 8:00 P. M.

November 13 — Tri County Medical Society — 4:00 P. M., Galesburg

November 18 — 7th District Post-Graduate Conference — Ottawa

October 14

SELECTIVE SERVICE MEETING

An interesting and unusual type of meeting will be held at Monmouth on Tuesday Evening, October 14, 1941, when Col. Paul G. Armstrong, Director of the Illinois Selective Service and Major Corwin S. Mayes, Chief Medical Officer of Selective Service will talk on the several phases of Selective Service.

Physicians and Dentists of Warren, Knox, McDonough and Mercer Counties including the personnel of the Medical Advisory Board No. 16 and of the five local draft boards in the four counties will be present.

The meeting has been arranged so that talks will be given by the two guest speakers, then a round table discussion of problems relative to examination of selectees, and the relationship between the local board examiners and the medical advisory board.

It is the desire of those arranging the meeting to endeavor in every way possible to see a reduction in the number of men approved by local examiners who are rejected at the induction station.

The meeting will be held at Hawcock's Cafe, Monmouth beginning at 6:00 o'clock with dinner, then the program immediately following.

5th DISTRICT
Post Graduate Conference
October 16, Lincoln
Lincoln State School and Colony
Dr. N. A. Balding, President
Logan County Medical Society, Presiding

12:30-1:30 — Luncheon — complimentary
1:30-2:00 — "Poliomyelitis" — Harry Leichenger, M. D. — Assistant Professor of Pediatrics, University of Illinois College of Medicine
2:00-2:40 — "Coronary Artery Disease" — Chauncey C. Maher, M. D. Assistant Professor of Medicine, Northwestern University Medical School
2:40-3:15 — Tour of Lincoln State School and Colony
3:15-3:45 — "Office Gynecology" — Herbert E. Schmitz, M. D. Chairman Department of Obstetrics & Gynecology, Loyola University School of Medicine
4:00-5:00 — Round Tables — led by
Gerald M. Cline, M. D. — Bloomington — Pediatrics.
Hermon Cole, M. D. — Springfield — Medicine — Gynecology
6:00-7:00 — Dinner
7:15-8:00 — "The Problem Child" — Bert I. Beverly, M. D. Assistant Professor of Pediatric Psychiatry Rush Medical College
"Surgery" —

4th COUNCILOR DISTRICT POST GRADUATE CONFERENCE
October 23, 1941
Dr. Paul P. Youngberg, President Rock Island County Medical Society, Presiding

1:00-2:00 P. M. — Luncheon, compliments of Rock Island County Medical Society
2:00-2:30 P. M. — "Common Diseases of the Skin and Their Management"
Cleveland J. White, M. D. — Assistant Professor of Dermatology Northwestern University Medical School
2:30-3:30 P. M. — "Heart Clinic" —
Robert S. Berghoff, M. D. — Clinical Professor of Medicine, Loyola University Medical School
Donald A. Hirsch, M. D. — Clinical Instructor of Medicine, Loyola University Medical School
3:30-4:00 P. M. —"Bowel Obstruction in the Newborn Infant"
Edwin M. Miller, M. D. — Clinical Professor of Surgery, Rush Medical College
4:00-5:00 P. M. — Round Tables — Dermatology, Medicine, Surgery
5:00-6:00 P. M. — Social Hour
6:00-7:00 P. M. — Dinner
7:30-8:30 P. M. — "The Duties of the Medical Officer" —
Ford K. Hick, M. D., Captain, General Dispensary, United States Army, Chicago

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AMERICAN CONFERENCE ON INDUSTRIAL HEALTH
November 5-6

Under the auspices of the American Association of Industrial Physicians and Surgeons the American Conference on Industrial Health will hold its Second Annual Meeting on November 5 and 6, 1941 at Chicago Towers, Chicago.

The opening session will be a symposium on the technical problems of industrial health on the basis that health supervision in industry involves the adjustment of the working environment to the employee; and the adjustment of the employee to the working environment. The afternoon session will be a symposium on the economics of industrial health, including organization and cost of a health service, and discussion on the value of industrial health service to the employer, the employee and the public. The second morning will be given over to a symposium on the social implications of industrial health. Sessions will close with a schedule of plant medical department inspections, by special arrangements with local industries.

8th COUNCILOR DISTRICT POST GRADUATE CONFERENCE
November 6, 1941
Hotel Wolford, Danville, Illinois

12:30-1:30 — Luncheon
1:30-2:00 — "Management of Prolonged Labor" — Frederick H. Falls, M. D.
2:00-2:30 — "Present Status of Effective Vitamin Therapy" — John R. Vonachen, M. D.
2:30-3:00 — "Office Gynecological Procedure or Treatment" —
3:00-4:00 — Heart Clinic with presentation of cases
4:00-4:30 — subject not definite
4:30-5:30 — Round Table group meetings
6:00-7:00 — Dinner
7:00-7:45 — "Medical Management of Upper Gastro-

Intestinal Tract Ulcer" — Ralph Brown, M. D.
7:45-8:30 — "Surgical Management of Upper Gastro-Intestinal Tract Ulcer" — Raymond W. McNealy, M. D.

PROGRAM
Sixty-seventh Annual Meeting
THE DISTRICT MEDICAL SOCIETY OF CENTRAL ILLINOIS
THURSDAY, NOVEMBER 6, 1941.
SPRINGFIELD, ILLINOIS

MORNING SESSION — St. John's Hospital
8:30 Springfield Tumor Diagnostic Service
(This conference only will be held at Springfield Hospital)
9:30 Clinicopathological Conference
Drs. H. Steen and Wm. DeHollander, St. John's Hospital
Drs. A. Vass and D. Sirca, Springfield Hospital
11:15 Cardiac Clinic
Dr. Fred M. Smith, Iowa City, Ia.
12:30 Luncheon
AFTERNOON SESSION — St. John's Hospital
1:30 Business Meeting
2:00 "The Basic Problems of Acute Appendicitis"
Dr. Frederick F. Boyce, New Orleans, La.
3:00 "Diaphragmatic Hernia"
Dr. Kenneth H. Schnepp, Springfield, Ill.
3:30 Intermission
4:00 "Encephalitis of Virus Origin"
Dr. H. J. Shaughnessy, Chicago, Ill.
4:45 "Non-specific Infections of the Urinary Tract"
Dr. Burle B. Madison, Springfield, Ill.
EVENING SESSION — Elks Club
6:00 Cocktail Party
Compliments Sangamon County Medical Society
6:30 Dinner — Elks Roof
8:00 "Certain Aspects of the Treatment of Cardiac Failure"
Dr. Fred M. Smith, Iowa City, Ia.
8:45 "Cancer of the Stomach"
Dr. Frederick F. Boyce, New Orleans, La.

TRI-COUNTY MEETING
November 13, 1941
Galesburg, Illinois

4:00 P. M. — "The Common Blood Diseases Seen in General Practice" — Howard L. Alt, M. D. — Assistant Professor of Medicine Northwestern University Medical School
5:00 P. M. — "Office Gynecology" — Ralph A. Reis, M. D. — Assistant Professor of Obstetrics and Gynecology, Northwestern University Medical School
6:00 P. M. — DINNER
7:30 P. M. — "The Clinical Significance of Hoarseness" — motion pictures in kodachrome of the various laryngeal conditions causing hoarseness. Francis L. Lederer, M. D. — Professor and Head Department of Laryngology, Rhinology and Otology University of Illinois College of Medicine.
8:30 P. M. — "Peripheral Vascular Disease" — Geza deTakats, M. D. — Associate Professor of University of Illinois College of Medicine.

The Thirteenth Annual William T. Belfield Memorial Lecture will be delivered on Thursday, October 16, 1941, at the Palmer House, at 8 P.M.

The lecture, "Evolution in Urology," will be delivered by Henry G. Bugbee, M.D., New York, New York.

MILITARY SURGEONS MEETING
Oct. 29th — Nov. 1st
Brown Hotel, Louisville, Ky.

Among the many medical meetings of this year is that of The Association of Military Surgeons of the United States to be held Oct. 29th - Nov. 1st at the brown Hotel, Louisville, Ky.

All members of the medical profession are invited to attend as guests and it is particularly hoped that as many members of the Medical Defense Committees as possible will come.

War medicine and surgery has changed considerably since the previous emergency. Mechanization of armies and air bombardments have created new and difficult problems in traumatic surgery and methods of treatments of wounds and extreme abrasions.

For every member of the profession who can be present at this meeting there will be something of special interest.

The session concludes with a mass review of Military Medicine and an inspection of Fort Knox.

MARRIAGES

PHILIP A. FUQUA, Joliet, Ill., to Miss Alice Ruth Campbell of Omaha, June 15.

SAMUEL PILCHMAN, Ladd, Ill., to Miss Adelyn Sylvia Baker of La Salle in Brooklyn, July 30.

G. ROGERSON WOLTMANN, M. D. of Chicago and Miss Betty Thomas of Urbana, Illinois were married on August 2, 1941.

DEATHS

EUGENE LESTER BAKER, Oak Park, Ill.; University of Illinois College of Medicine, Chicago, 1925; major, 108th medical regiment, Illinois National Guard, Camp Forrest, Tenn.; aged 42; was killed, July 20, in an automobile accident.

ARTHUR ROOSEVELT BOWLES, Chicago; Yale University School of Medicine, New Haven, Conn., 1931; received his bachelor's degree from Columbia University, New York, in 1927; served a two year medical internship at the New Haven Hospital and a one year pediatric internship at the Strong Memorial Hospital,

Rochester, N. Y.; during the winter of 1934-1935 studied hospital administration at the University of Chicago on a Rosenwald fellowship; from 1935 to 1937 was assistant director and during 1937 and 1938 acting director of the Grasslands Hospital, Valhalla, N. Y.; since October 1939, hospital inspector on the staff of the Council on Medical Education and Hospitals of the American Medical Association; aged 35; died, September 3, in the City Hospital, Indianapolis, of malignant hypertension with nephritis.

HERSCHEL VICTOR BRUNKER, Casey, Ill.; Bennett College of Eclectic Medicine and Surgery, Chicago, 1915; served during the World War; aged 53; died, July 7.

JOSEPH SWISHER CUNNINGHAM, Gibson City, Ill.; *Illinois Medical College, Chicago, 1902*; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1907; member of the Illinois State Medical Society; aged 67; died, August 21.

CHARLES SUMMER DAVIS, Champaign, Ill.; Eclectic Medical Institute, Cincinnati, 1890; served during the World War; aged 75; died, July 31, of heart disease.

ERNEST CHRISTIAN DAY, Chicago; Rush Medical College, Chicago, 1936; member of the Illinois State Medical Society; captain, 108th medical regiment, Illinois National Guard, Camp Forrest, Tenn.; aged 37; was killed, July 20, in an automobile accident.

ROY HENRY GARM, Beardstown, Ill.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1901; served during the World War; aged 62; died, July 23, in the Norbury Sanatorium, Jacksonville, of cerebral hemorrhage.

PHILIP CONSTANT GOERGEN, Chicago; Loyola University School of Medicine, Chicago, 1919; examining physician for the draft board; aged 56; died, July 2.

PHILIP GRIESBAUM, Lebanon, Ill.; Washington University School of Medicine, St. Louis, 1910; member of the Illinois State Medical Society; president of the high school board of education; aged 57; died, July 29, in Jacksonville.

WALTER MARION HAGGETT, Chicago; College of Physicians and Surgeons, Keokuk, Iowa, 1887; member of the Illinois State Medical Society; aged 83; died, July 17.

HORACE BLAKE HANNON, Chicago; College of Physicians and Surgeons of San Francisco, 1903; aged 66; died, August 8.

JOHN I. HIGGS, East St. Louis, Ill.; Missouri Medical College, St. Louis, 1885; aged 81; died, July 15, in Sparta of coronary occlusion.

FRANK BRENNAND KIRBY, Evanston, Ill.; Jefferson Medical College of Philadelphia, 1902; aged 63; died, August 20.

LEWIS HERTZ LIPPMAN, Chicago; Chicago College of Medicine and Surgery, 1913; member of the Illinois State Medical Society; aged 53; died, August 9, in St. Mary's Hospital, Kankakee, Ill., of coronary thrombosis.

WILLIAM HINKLE MASSEY, Surgeon Lieutenant Commander, United States Navy, retired, Peoria, Ill.; Cooper Medical College, San Francisco, 1912; entered the navy Aug. 17, 1914 and retired June 14, 1923 for incapacity resulting from an incident of service; served during the World War; aged 55; died, August 16.

MARY R. MCCONAHY, Evanston, Ill.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1890; aged 101; died, July 26, of arteriosclerosis.

ALICE MAY PARSONS, Chicago; Rush Medical College, Chicago, 1933; aged 44; died, August 12, in the *Chicago Memorial Hospital of chronic rheumatic* heart disease.

WILLIAM BUCKLEY PECK, Freeport, Ill.; Rush Medical College, Chicago, 1897; fellow of the American College of Surgeons; founder and managing director of the Inter-State Postgraduate Medical Association of North America since its inception in 1916; formerly on the staffs of St. Francis Hospital and the Deaconess Hospital; aged 70; died, August 20, of coronary heart disease.

KATHERINE BRAINERD RICH, Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1901; aged 77; died, August 14, in Henrotin Hospital of cerebral hemorrhage.

HYMAN B. SHAPIRO, Chicago; Rush Medical College, Chicago, 1903; aged 63; died, June 11, of gastric hemorrhage.

CHARLES JOSEPH SWAN, Evanston, Ill.; Hahnemann Medical College and Hospital, Chicago, 1890; Northwestern University Medical School, Chicago, 1909; fellow of the American College of Surgeons; served during the World War; formerly on the staff of the Evanston Hospital; aged 76; died, August 6, of coronary occlusion.

JOHN LINTON JOSEPH VALLELY, Chicago; Northwestern University Medical School, Chicago, 1910; on the staffs of the Augustana and Edgewater hospitals; aged 73; died, August 7, of coronary sclerosis.

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